New Orleans Medical
and
Surgical Journal

Vol. 99 DECEMBER, 1946 No. 6

INFECTIOUS HEPATITIS IN CHILDREN

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The high incidence of acute liver disease in the armed forces of the United States during World War II has stimulated marked interest in this subject. The outbreak of jaundice of mysterious type in the training camps in 1942, later demonstrated to be due to homologous serum was followed by epidemics of acute infectious hepatitis, especially in the Mediterranean theater of operations.[1] The establishment of the Hepatitis Commission, with small teams of research workers, resulted in the clarification of many details concerning these infections. The size of the clinical problem is evident in the report of one group from the Mediterranean theater which conducted a study of 1172 cases of hepatitis, in addition to special surveys on several thousand others. Two of these investigators had previously made extensive clinical and laboratory studies of 2000 trainees who had received yellow fever vaccine, of whom 700 were hospitalized with jaundice.[1]

Prior to these investigations, a considerable amount of confusion existed, despite a few reports which were pointing toward the present conception of acute hepatitis. The severe hepatitis due to *Leptospira icterohaemorrhagiae* had been differentiated by Weil in 1886 and shown to be of spirochetal etiology by Japanese investigators[2] in 1914. Homologous serum jaundice had been described by Findlay and MacCallum[3] in 1937 but was not generally recognized. Acute catarrhal jaundice was still considered to be different from acute infectious hepatitis, and to represent an ascending cholangitis, with jaundice resulting from mucus obstruction of the bile ducts, although Rich[4] and others had presented evidence that "catarrhal jaundice" was due to hepatitis of systemic origin and Blumer[5] had believed catarrhal jaundice to be the sporadic form of epidemic or infectious hepatitis. The frequency of residual liver damage after infectious hepatitis and the important role of dietary factors in preventing liver damage were not generally known.

Extensive investigations of homologous serum hepatitis and infectious hepatitis[6, 7] have established a number of salient facts concerning their etiologic agents, despite the failure of attempts to isolate, identify or grow causative organisms. The agents of both diseases are filterable viruses, are resistant to heat at 56 degrees C, ,survive for long periods of time at room temperature and in frozen feces or urine, survive exposure to 1 :2000 merthiolate and are not affected by the ordinary methods of chlorination. Animal inoculations are unsuccessful, lesions are not produced in the chick embryo and inclusion bodies have not been found. They produce similar manifestations in humans, and pathologic changes of inflammation and necrosis of liver cells without involvement of the larger bile ducts are identical. Both agents are present in the blood at some time during the respective

disease. The fatality rates are about the same (0.2 to 0.3 per cent) and fatal outcome in each group is preceded by marked evidences of central nervous system involvement.

Certain differences are noted. In infectious hepatitis, the incubation period is usually less than 40 days, while in serum hepatitis it is 60 to 150 days. The onset of homologous serum hepatitis is usually afebrile and the temperature usually does not exceed 100 degrees, whereas in infectious hepatitis, it usually exceeds 100 degrees F. Contact cases are rare in serum hepatitis, more common in infectious hepatitis. Neefe, Stokes and Gellis[7] present evidence that each disease produces homologous immunity, but not a cross immunity. Transmission in serum hepatitis is effected by injection of infected blood or serum, as in transfusions and other intravenous medications, or material containing serum as a stabilizing agent, as happened in the instance of yellow fever vaccine; infectious hepatitis is transmitted largely by ingestion of materials contaminated with feces.

The latter point was established by general observations that epidemics of the disease are more frequent under crowded, unsanitary conditions, and specifically by the epidemiologic studies of Neefe and Stokes[8] during an epidemic which occurred in a summer camp near Philadelphia in 1944, during which 350 (61 per cent) of a total of 572 boys, girls and adult attendants developed hepatitis. Epidemiologic observations and transmission experiments in human volunteers provided evidence that the causative agent was not transmitted by air, fomites, biting insects, flies, food, milk or water from the lake nor directly from infected persons, but was transmitted by water from a well which contained a transmissible agent which was apparently the same as that excreted in the feces of infected persons.

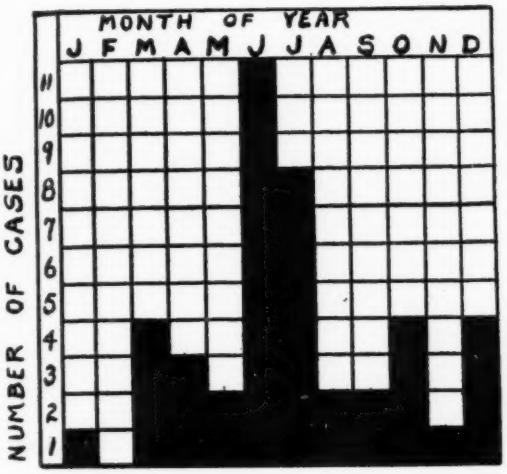
INCIDENCE OF HEPATITIS

A number of reports have indicated a marked increase in the incidence of virus gastroenteritis in the civilian population of the United States during the war years. In our area of North Louisiana this has been paralleled, to a lesser degree, by an apparent increased incidence of acute hepatitis. Our Clinic records show only one child with the disease during the ten years prior to 1941, with an incidence of 41 since that time (table 1). The increased incidence has been particularly noted during the past three years, with 16, 6 and 16 cases occurring during 1944, 1945 and 1946, respectively. In several areas, the disease appears to be endemic. In the town of Mansfield, a parish seat of about 5,000 population situated 40 miles south of Shreveport, ten to twelve children between the ages of one and ten years have had the disease since 1944, only one of whom is included in our Clinic series. Two other towns, Arcadia, La., and Atlanta, Texas, are known to have had four to six cases each. At the Shreveport Charity Hospital during the winter of 1944-1945, seven nurses and five physicians had acute hepatitis during a period of two months. One of the physicians still has chronic hepatitis resulting from this illness.

TABLE 1
CASES OF INFECTIOUS HEPATITIS BY YEARS

	1934	1941	1942	1943	1944	1945	1946	Totals
Icteric	1	1	2	0	9	3	4	20
Non-icteric	0	0	0	0	7	3	12	22
Totals	1	1	2	0	16	6	16	42

A study of our series of 42 cases shows that 22 lived in Shreveport while the remaining 20 came from 18 different communities in North Louisiana, East Texas and South Arkansas within a radius of 100 miles. Ten of these children were referred by the home physician, while the remainder had previously been under our care. This might indicate that a number of other cases of the disease had been treated by the home physicians in the respective communities, and reports from some of the families indicated that such was the case. In 1946, twelve of the individuals (ten children, two mothers of affected children) lived in the same general area in Shreveport and became ill during the months of May through July. Half of them had possible direct contacts, through school, play or family associations, but all other cases appeared to be sporadic.



The seasonal incidence (fig. 1) shows a spread throughout the year, with greatest prevalence during the summer months.

A survey of age incidence (table 2) shows that almost half (20) of the affected children were between five and ten years of age, 15 were between one and five years, five were between ten and fifteen years and two were adults (mothers). No infants under one year were seen with the disease. Since 30 of these cases occurred in our regular clientele, interpretation of these figures in accordance with our case load distribution by ages is necessary. Recent figures for the Study of Child Health Services show that the pediatric case load is greater in the one to five year group than in the group above six years; hence the true incidence of hepatitis in older children is probably even higher than indicated by our present series.

TABLE 2
CASES OF INFECTIOUS HEPATITIS BY AGE

	1-5 yrs.	6-10 yrs.	11-15 yrs.	Adult
Icteric	6	11	3	0
Non-icteric	9	9	2	2
Totals	15	20	5	2

HOMOLOGOUS SERUM HEPATITIS

The possibility that several of our cases of hepatitis may represent homologous serum hepatitis must be considered. In February and April, 1944, two boys of six and seven years received intravenous scarlet fever convalescent serum, 20 c.c. each from the same lot, for treatment of this disease. After intervals of 93 and 60 days respectively, they became ill with acute hepatitis, icteric in one instance, non-icteric in the other. No other children given the serum developed jaundice and the serum center[9] reports that no other cases of homologous serum hepatitis developed from this lot of serum, so that the hepatitis in these two instances was probably coincidental. One other child in our series developed hepatitis 189 days after receiving a transfusion, but this would be an extremely long incubation period; furthermore, this donor has no history of hepatitis and other transfusions from the same donor were not followed by jaundice. No other children in our series had a history of serum or blood administration within a year prior to inception of the hepatitis.

SYMPTOMS AND CLINICAL COURSE
A: ICTERIC CASES

Twenty children developed clinical icterus; an equal number of the children and the two mothers had non-icteric hepatitis. Those who developed jaundice were usually sicker, the jaundice being preceded by a pre-icteric stage of fever, ranging from 99 to 104 degrees F. for a period of two to five days, anorexia, malaise and usually pronounced abdominal symptoms, nausea, vomiting and upper abdominal pain being the more frequent. Occasionally constipation loose stools, coated tongue or general abdominal soreness were mentioned and the stools were noticeably light colored in six instances. Other symptoms were headache, restlessness or irritability, epistaxis, dark colored urine, and weight loss. In two instances, chills accompanied the initial fever and malaria was suspected, but no parasites were found and therapeutic trial was without effect.

While fever usually had subsided in from one to several days before jaundice appeared, low grade fever sometimes persisted. In three instances, the children had acute upper respiratory infections beginning ten days to two weeks before inception of the jaundice, with fever for several days, then an afebrile period of a week to ten days during which the respiratory symptoms persisted, together with malaise and increasing

abdominal symptoms before icterus appeared.

In four of the children, icterus was noted only in the sclerae, while in the remainder the skin and sclerae were icteric in varying degree. A "sallow" color was often noted by the parents before clinical icterus became apparent. Itching or scratch marks on the skin were present in three instances and two children had evanescent skin rash on the first febrile day. The duration of the jaundice averaged ten days to two weeks, although three weeks was not unusual and two children were slightly jaundiced for a month. Anorexia or lack of vigor sometimes persisted for a week or two after icterus cleared. In other cases, the children were jaundiced for only a week and seemed to recover very rapidly. The duration of the entire illness in the icteric group varied from ten days to two months, with the usual course being two to three weeks.

SYMPTOMS AND CLINICAL COURSE
B. NON-ICTERIC CASES

The clinical symptomatology in the non-icteric group was milder. Fever was often absent or unrecognized, although recordings of 99 to 101 degrees F. for periods of one to three days were not unusual and two children had temperatures of 103 to 104 degrees. Malaise, fatigue, irritability, dizziness, headaches and vague abdominal complaints were the rule, but some of the patients had more severe gastrointestinal symptoms — vomiting, upper abdominal pain, loose stools. Acholic stools were noted in five instances. The milder cases suggested the clinical picture formerly called "biliousness." Although no clinical icterus was in evidence, the skin was noted to be "sallow" or pale in about half of the cases, and a history of itching or scratching was secured several times.

Antecedent illnesses included tonsillitis, upper respiratory infection, measles and— in two instances — acute gastroenteritis (virus type), all occurring within a month before the hepatitis. Two patients referred from other towns had histories of siblings ill several weeks before, one diagnosed as virus pneumonia, the other as acute upper respiratory infection, with clay-colored stools noted during the illness.

PHYSICAL FINDINGS

The findings on examination were often disappointingly few, except for the skin and scleral coloration. Slight adenopathy, especially cervical, was noted rarely. The liver was perceptibly enlarged in 10 of the icteric cases and four of the non-icteric, noted as two to three finger breadths below the costal region and tender in a few instances. Soreness or mild rigidity of the upper abdominal muscles was noted several times. The spleen was palpable in seven instances, extending one to two finger breadths below the costal margin. Slow heart rate, skin rash and scratch marks on the skin were other occasional findings. When vomiting had been excessive, mild dehydration, reddened or cracked lips and acetone odor were present.

LABORATORY FINDINGS

In view of the repeated demonstration [1, 8] that 50 per cent or more of the individuals infected with acute hepatitis in an epidemic never develop visible jaundice and that the symptom complex in the absence of jaundice may simulate a number of other diseases, we were interested in discovering which laboratory procedures are reliable and adaptable for use in our clinic. No single test is diagnostic of this disease, but correlation of the routine blood examination, urine tests and two or three selected liver function tests with the clinical picture will at least give evidence of liver dysfunction strongly presumptive of infectious hepatitis.

Blood counts: complete blood counts were made one or more times in 28 of the 42 patients we have seen. The red blood cell counts and hemoglobin estimations were within the normal range, unless the child had antecedent anemia, and the erythrocytes showed nothing unusual. This finding is in contrast to the pallid appearance of the skin in non-icteric individuals. The leukocyte count varied considerably, even in uncomplicated cases, with a range from 3,200 to 15,000 cells per cu. mm. However, moderate leukopenia was the general rule, with 17 of the 23 uncomplicated cases hav-

ing counts of 7,000 or below. Distribution of the leukocytes also varied considerably, with neutrophiles varying from 13 to 75 per cent, lymphocytes from 21 to 66 per cent, monocytes 1 to 15 per cent and eosinophiles 1 to 11 per cent.

The striking finding on examination of blood smears was the appearance of the lymphocytes, an appearance first noted in the instance of children with acute (virus) gastroenteritis during the epidemic of 1943-1944. For a time the technicians called us to the laboratory almost daily to discuss the unusual lymphocytes, which we came to speak of as having the "virus look", especially after we noticed the same type of cells in acute hepatitis during the past two years. These lymphocytes appear slightly young, with azure stained granules in the cytoplasm, the nucleus often indented and having clumped chromatin, and vacuoles often appear in the nucleus or cytoplasm. Barker, Capps and Allen[1] mention that 5 to 20 per cent of the lymphocytes in smears from their series of infectious hepatitis were atypical, similar to those of infectious mononucleosis, and one of these authors[10] later informed us that similar lymphocytes were frequent in studies of acute virus gastroenteritis in army personnel. No published report of this observation has come to our attention. Undoubtedly the abnormal lymphocytes seen in these three diseases are similar, although in our experience the changes in acute mononucleosis are more striking, and the lymphocytes seen in hepatitis and gastroenteritis of virus origin do not often have the typical cartwheel arrangement of nuclear chromatin.

Urine examination: The urine is usually dark colored during the period of initial illness, well before jaundice occurs. In the non-icteric, this dark color may last for only a few days, but in sicker patients, it persists for some time. A trace of albumin is not infrequent. The tests of importance, however, are those for bile salts and bilirubin, which have been positive in all of our cases, icteric and non-icteric. We have found that as the child improves, the jaundice clears, then within a few days the urine bilirubin test becomes negative, but a positive bile test is likely to persist several more days. Liver function tests are likely to show activity for quite a period of time after all urine tests are normal, so that positive tests for bile or bilirubin in the urine are of importance in diagnosis but not in judging complete recovery.

The bile tests which we have used are the nitric acid ring test or old nitric acid saturation of filter paper, to which a drop of urine is added. Surveys which we have made and will report in detail later, however, demonstrated that these tests are not as specific and apparently are subject to more variation in interpretation than the bilirubin test. Even with the latter, proper technic seems to be of importance to avoid misinterpretation. We first used Myers'[11] modification of Franke's methylene blue test for bilirubin, in which two drops of Loeffler's methylene blue are added to 10 c. c. of urine. If a green color results, the test is considered positive and is repeated with increasing dilutions of urine until the dilution is reached in which two drops of the reagent produces a blue color, thus giving a roughly quantitative test. In surveys of clinic patients and hepatitis contacts with this test, irregular results and high percentage of positives led to the adoption of a similar test described by Gellis and Stokes[12] which in our laboratory has seemed much more satisfactory. This technic is as follows: to 5 c. c. of urine (preferably prebreakfast specimen) is added two drops of an 0.2 per cent aqueous solution of methylene blue chloride. If a green color results, more drops of the methylene blue are added and the last drop required to convert the green color to blue is recorded. If more than five drops are required, the test is considered positive. Gellis and Stokes found close correlation between results of this test and the icterus index in 77 cases of hepatitis, with positive methylene blue tests preceding clinical icterus by one to six days. Although less sensitive than the Harrison spot test and open to some objection in that the green color is due to a blend of blue and yellow colors and not to a chemical combi-

nation between bilirubin and methylene blue,[13] its simplicity makes it valuable for clinical use in any laboratory.

Liver function tests: There are numerous tests of liver function and obviously the clinician will find it neither expedient nor necessary to secure a large number of these. It seems better, therefore, to attempt to evaluate them in the light of information secured and adaptability to clinical use. For example, the icterus index is largely confirmatory and of some value in following a case closely, but not very important in diagnosis, since the degree of clinical jaundice and the icterus indices are closely correlated. In our series, for example, values of 14 to 45 units were found in icteric cases; four to 18 in the non-icteric (table 3). The van den Bergh is of more value, especially in differentiating hepatitis from the icterus of hemolytic blood dyscrasias, as a positive direct reaction usually occurs in the former but not in the latter. A prompt direct reaction was secured in eight to ten patients in which we used this test, including the non-icteric (table 3). Quantitative serum bilirubin was in excess of 2 mgm. in the icteric patients but in six non-icteric patients it was below this figure. Serum protein determinations in two instances showed lowered albumin and elevated globulin values, but these determinations are time consuming in view of limited information secured.

Numerous other tests—bromsulfalein retention, alkaline phosphatase, hippuric acid excretion, glucose tolerance, cephalin cholesterol flocculation test of Hanger, thymol turbidity, serum cholesterol, serum amylase and prothrombin time, to mention only a few—have been used by investigators. All liver functions may be disturbed in varying degree during acute hepatitis, even in the mildest, non-icteric form. We have used the flocculation test of Hanger, as reports had indicated its adaptability to clinical use and comparative reliability as an indicator of liver damage. All of our icteric cases which were tested and eight of the 12 non-icteric cases showed 2 to 4 plus Hanger readings (table 3). In the large series of cases reported by the Hepatitis Commission[1] Hanger tests were positive in 50 to 75 per cent during the prodromal stage, in 90 per cent during the acute icteric stage, and in 53 per cent of the non-icteric cases. In their series, the bromsulfalein dye retention proved to be the most reliable test, while others consider the thymol turbidity test to be more sensitive.[14]

TABLE 3
LABORATORY FINDINGS IN 12 CASES OF NON-ICTERIC HEPATITIS

Patient	Age in years	Leukocyte count Cells per cu. mm.	Percentage of lymphocytes	Icterus index	Van den Bergh qualitative	Serum bilirubin (mgm. per 100 cc.)	Hanger's cephalin cholesterol flocculation	Urine bile	Urine bilirubin
M. M.	11	3,950	59				2+	Pos.	Pos.
K. M.*	42			5.8	Pos.		3+	Pos.	Pos.
E. W.	10	4,100	54**	5.0	Pos.	1.4	4+	Pos.	Pos.
D. W.*	41			8.0	Pos.		Neg.	Pos.	Pos.
J. M.	10	21,000†	35**	8.5	Pos.	1.8	Neg.	Pos.	Pos.
M. A. M.	11	5,000	52	5.0			2+	Pos.	Pos.
R. L.	4	6,000	66**		Trace		2+	Pos.	Pos.
C. E. A.	6	3,200	63**		Trace	0.26	3+	Pos.	Pos.
B. S.	6				Pos.	0.76	Neg.	Pos.	Pos.
J. S.***	4			4.7	Neg.	0.23	Neg.	Pos.	Pos.
J. B.	3				Pos.	0.32	3+	Pos.	Pos.
J. G.	5	8,900	18	18.8	Pos.			Pos.	Pos.

* Mothers of M. M. and E. W. respectively.
** Atypical lymphocytes seen on blood smears.
***Sister of B. S.
†Patient also had acute maxillary sinusitis.

PROPHYLAXIS AND TREATMENT

Since this disease is demonstrably contagious, care should be taken in disposal of excreta from the individual case and children should be kept away from the infected individual. In the presence of epidemics or in endemic foci, food and water supplies should be carefully checked. Individuals with histories of jaundice should not be used as donors for transfusions or sera.

Stokes and Neefe[15] and Gellis et al.[16] have demonstrated, in the presence of epidemics of infectious hepatitis, that the administration of gamma globulin to contacts reduced the incidence of the disease by 60 to 80 per cent, and modified the illness in the remainder. The dosage of gamma globulin used was 0.15 c. c. per pound body weight in children, 10 c. c. in adults, in a single injection.

In treatment of the active case, two considerations of prime importance are rest and diet. Experiences in the armed forces[1] showed that early and complete bed rest for an adequate period of time decreased the severity and reduced the incidence of delayed convalescence. Bed rest should continue until symptoms have cleared, the liver has returned to normal size and is not tender, the urine is clear of bilirubin and one or more of the liver function tests (Hanger, bromsulfalein, van den Bergh, thymol turbidity) are normal. In case of recurrence of symptoms, as happened in three of our cases, immediate resumption of bed rest is essential.

The diet should be high in protein and carbohydrate, low in fat. The experimental work of Miller, Whipple, Györky and others[17, 18, 19, 20] indicates that a high protein diet is far more protective to the liver than a high carbohydrate diet, in fact, that the latter is probably of value chiefly because of its protein sparing effect. Further study indicates that the sulfur-containing amino acids, methionine especially, or cystine plus chlorine, are the particular substances in the high protein diet which protect the liver. Foods reported to be rich in methionine are casein, serum albumin and egg white.

Eddy[21, 22] found methionine to be valuable in treatment of toxic hepatitis of chemical origin and due to his efforts methionine has been available locally. We have used it in several cases of infectious hepatitis, in dosage of two to three grams daily, apparently with good results. Prior to that time we had advised high proteins, especially skimmed milk (casein), with choline.

Due to fat restriction, extra supplies of fat soluble vitamins are desirable, also vitamin B complex in moderate amounts. Ample fluids should be given in the acute stage and, in the severely ill, intravenous fluids may be essential.

SUMMARY

1. The high incidence of acute infectious hepatitis in the armed forces has been paralleled by increased incidence in the civilian population in this area during the past three years.

2. Non-icteric cases of this disease appear to equal or exceed the icteric. They exhibit the same general symptom complex in milder form and if suspected may readily be demonstrated by proper diagnostic procedures.

3. Tests which are readily available to any good clinical laboratory are adequate for diagnosis of acute hepatitis. Those which we have found to be of especial value are: Leukocyte count with differential study, urine tests for bile and bilirubin (especially methylene blue test for bilirubin), qualitative van den Bergh, quantitative serum bilirubin and Hanger's cephalin cholesterol flocculation.

4. Abnormal lymphocytes found in this disease are apparently identical with those found in acute (virus?) gastroenteritis and very similar to those found in infectious mononucleosis.

5. Important principles of treatment include adequate and prolonged rest and high protein, low fat diet. The amino acid methionine is the important liver-protecting constituent of the dietary protein.

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ACUTE DIARRHEA IN CHILDREN

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Diarrheal disorders are widespread and constitute in the summer months one of the major causes of illness and infant mortality. This is particularly true in the Southern tier of states. The child brought to the physician with a chief complaint of diarrhea may be suffering from any one of a large group of diseases. The recognition of the etiologic agent and the treatment of the acute illness are the two important problems confronting the medical practitioner.

The accurate diagnosis of these diarrheal disorders is a problem for the laboratory, since it has been shown repeatedly that clinical findings in an individual case can do little more than suggest the etiological agent. There is one point, however, which is of some practical value to the physician in the South. Usually a diarrhea which is sufficiently severe to cause the mother to consult a physician is caused by one of two large groups of intestinal pathogens, the *Shigella* and *Salmonella* groups.[1] In all parts of the country that have been studied, the *Shigella* group predominates, but the relative importance of the two varies considerably.[2] In some areas the *Salmonella* infections have constituted less than one per cent of the endemically occurring cases, whereas in others, specifically in the New Orleans area, the *Salmonella* group has been isolated from as high as 25 per cent of the cases admitted to a hospital service. For practical purposes, in the absence of laboratory facilities, the physician can safely assume that something over three-fourths of his cases of acute diarrhea are due to an organism belonging to one of the above groups, and it is therefore good practice to assume that all of these disorders are infectious until proved otherwise. This is particularly important when there are other young children in the family.

DIAGNOSIS

As stated previously, the problem of diagnosis is basically a laboratory problem, and in the last few years this has been tremendously simplified. The smallest laboratories can now do satisfactory enteric diagnostic work if they will avail themselves of the newer tools. Culture media are available in dehydrated form which permit preparation of fresh media on a few minutes' notice. They are specifically designed

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to promote the growth of enteric pathogens, and at the same time inhibit those nonpathogenic organisms which ordinarily are present in large numbers in any fecal specimen. The best medium for the purpose is called SS (*Shigella-Salmonella*) agar. For accurate diagnostic work, it is essential that this medium or a similar one be used.[3]

The selectivity of these media has greatly simplified the obtaining of necessary diagnostic specimens. When using older types of media, it was necessary to have someone obtain a specimen of the freshly passed stool, place it in a container and send it, often with considerable delay, to a selected laboratory. A considerable change in fecal flora usually took place under these circumstances before the laboratory had any opportunity for examining the specimen. By using the new media, it is now possible to obtain a specimen in much the same way as doctors have used for many years in the diagnosis of diphtheria. The media can be used in the office, or in the home, the specimen being obtained at the time of consultation. An ordinary cotton tipped applicator is inserted into the rectum of the patient. This is easily accomplished if care is taken to spread the buttocks as widely as possible before inserting the swab. A sufficient amount of feces will adhere to the cotton, and then the swab is used to plate the material directly on the culture medium. The entire surface of the plate should be covered, using a painting motion. This method has been used successfully in both hospital and private practice for the past four years. In one hospital where the routine practice of culturing each case on admission was in effect, the shortening of time required for a laboratory report was spectacular. It was found that when the rectal swab was taken and plated on admission of the patient, it was possible for the laboratory to give a preliminary report, on the average, 36 hours later. When the attending physician simply ordered a stool specimen to be sent to the laboratory, the average interval between admission and reporting was seven and a half days. When one considers the usual short duration of most of these infections, it is little wonder that physicians have taken very slight interest in enteric cultural work. The patient was either dead or recovered before the doctor had any report as to the results of the culture. By using the simplified method, however, a report can usually be obtained during the acute phase of the illness and at a time when it can probably influence management of the case. This will be brought out in more detail when specific therapy is discussed.

The whole field of enteric bacteriology is a complicated one, and the physician in practice cannot be expected to acquaint himself with all newer developments as they occur except in so far as they have practical application to the treatment of his cases. For this reason, we are not discussing the many different types of organisms which may be encountered in the course of a summer's work. For example, in one area in four months' time we isolated some eight different types of *Shigella* and 22 different types of *Salmonella*. This information is important to the epidemiologist in his search for sources and modes of spread of these infections. At our present stage of knowledge, however, there is little or no practical value to the physician in identification of these organisms beyond the particular genus to which they belong. The genus of the pathogen present is of significance because: (1) the *Genus Shigella* is susceptible to sulfonamide therapy whereas the *Genus Salmonella* is not; (2) the *Shigella* group is for practical purposes a human pathogen and lower animals are not hosts to this parasite. On the other hand, the *Salmonella* group are widespread in the lower animals. This has a considerable influence on any investigation designed to determine the source of an infection.

In summary, the laboratory tests outlined above, namely isolation and identification of the etiologic agent, are the only ones of practical diagnostic value. Agglutination tests have been used, but they, at best, could only give a retrospective diagnosis, and recent studies have indicated that in the individual case it is impossible reliably to

interpret positive or negative findings.[4] Complement fixation and other similar tests have not been developed.

When a satisfactory laboratory test has been performed with negative results, other possible causes of the diarrhea should be considered. Usually, however, in the acute illness found negative on culture, the duration is short and the patient's recovery is prompt, with little or no assistance on the part of the physician. The cause in these cases may be extremely variable, but the treatment is essentially the same for all.

TREATMENT

The major problem in the management of diarrheal diseases of children is treatment of the severe dehydration which frequently occurs in these patients, especially in infancy. In addition, acidosis is almost invariably present in these dehydrated infants and must be treated specifically. This problem is greatly simplified if laboratory facilities are available. The critically ill patient should have the CO_2 combining power of the blood determined immediately. In severe acidosis this figure is below 30 volumes per cent and one of the following solutions is indicated to correct the acidosis. Roughly, 1 c. c. per pound of body weight of either one sixth molar sodium lactate, or 1.5 per cent sodium bicarbonate is needed to raise the CO_2 combining power one volume per cent. Thus by subtracting the determined level from the normal of approximately 55 volumes per cent, the necessary number of c. c.'s per pound is obtained for either solution. At least half of the required amount should be given immediately by intravenous injection, and the remainder may be given subcutaneously or intraperitoneally. Dehydration is further combatted by the use of Ringer's solution and 6 per cent dextrose solution in approximately equal quantities. These solutions may be given intravenously or subcutaneously, and higher concentrations of dextrose are possible by vein if given slowly. The use of dextrose solution avoids an over-supply of sodium chloride, and in addition, it is particularly valuable in treating the ketosis and starvation which may be present. A total of 75 c. c. to 150 c. c. of fluid per pound of body weight are needed in the first day in severe dehydration and acidosis.[5]

The intravenous drip method of administration is particularly valuable in these cases of severe fluid loss. In the early stages of administration gravity flow through a 22 to 24 gauge needle may be permitted and after approximately half of the needed volume has been administered the rate of flow is cut to approximately 5 to 10 drops per minute. Where the drip method is not possible, single intravenous injections of 10 to 30 c. c. per pound may be given over a 20 to 30 minute period. This dose may be repeated every four hours until the dehydration is overcome. A large volume can be given subcutaneously even in small babies when the dehydration is marked. This method is frequently the most convenient for the practitioner who has to handle these patients in the home. Also where vomiting is not marked, much of the necessary fluids can usually be given by mouth if care and patience are exercised.

When the laboratory facilities for CO_2 determination are not available, some arbitrary rule of thumb must be used. It has been found that severe acidosis can usually be overcome by 15 c. c. per pound of one-sixth molar sodium lactate solution or 1½ per cent sodium bicarbonate solution given intravenously, followed by an equal amount administered subcutaneously. Ringer's and dextrose solutions are then given in the same manner and quantity described above.

When the fluid and electrolyte balance has been reestablished, about 60 c. c. of fluid per pound are necessary daily to maintain hydration in the normal infant. With continuing diarrhea, more than this is required, as well as approximately 15 c. c. per pound of one-sixth molar sodium lactate, to prevent the recurrence of acidosis. Ketosis may be avoided by 0.5 gm. of dextrose per pound of body weight each day.

The cases of diarrhea which fail to do well under the above regime may be suffering from a lack of potassium. This has

been brought out by the investigations of Darrow and his co-workers,[6] and a new solution designed to take care of this deficiency is now being investigated.[7] It promises to be of great value and probably will be standardized in the near future. At present, however, it is not generaly available.

When dehydration and acidosis are moderate, lactate-Ringer's is the solution of choice. This may be given orally except in cases of vomiting and in infants when parenteral injection is necessary because of the rapidity with which a dangerous condition may develop. It is much easier to *prevent* severe acidosis than to treat it once it has arisen. The mild cases are treated satisfactorily by forcing fluids, water or normal saline, by mouth.

In cases of diarrhea with excessive vomiting, there is considerable loss of chloride ions from the body. This lack is usually adequately made up by chlorides in the solutions given for dehydration. Ringer's solution is especially good and may even be tried by mouth in small quantities, one teaspoonful every fifteen minutes, and gradually increased as tolerated.

After body fluids have been restored, a whole blood transfusion, concentrated plasma, or amino acid hydrolysates may be extremely valuable. This is particularly true in the poorly nourished patient whose serium protein is very low. This treatment should be repeated as necessary, whenever recovery is slow; and in such cases, parenteral vitamin administration also is helpful.

Various sulfonamide preparations have been found to be effective in the treatment of *Shigella* infections. Since a high percentage of severe cases are caused by a member of this group of organisms, the use of a sulfonamide preparation is often indicated at once, even before a definite diagnosis is finally established.

Absorbed sulfonamides have been shown to be most effective, and of these, sulfadiazine, sulfapyrazine and sulfathiazole have been shown to be effective in the order named.[2] The dose should be approximately ¾ to 1 grain per pound of body weight daily. It is given in divided doses four to six times per day. Usually to insure more rapid therapeutic levels of the drug, the initial dose is doubled. When stool cultures are obtained, this therapy should be modified to conform with the findings. For the *Shigella* group of organisms, the sulfonamide should be continued until two negative cultures have been obtained, or for seven days, whichever comes first. Occasional organisms which have not responded by the end of seven days to such therapy are usually resistant strains, and continued administration of the drug is not beneficial. Since the *Salmonella* group do not respond to these drugs, isolation of this organism is an indication to stop sulfonamide therapy. A negative cultural result may be due to technical difficulties and in such a case the physician should be guided by the clinical results obtained up to that time. If the patient is unable to take medication by mouth, 0.5 per cent sodium sulfadiazine may be given parenterally along with other fluids. Dose schedules remain the same. Therapeutic levels of sulfadiazine are present in the stools within a matter of hours after parenteral administration. When using these absorbed sulfonamides, particularly sulfadiazine, it is important that the acidosis be vigorously combatted, since an alkaline urine is advisable for prevention of urinary complications.

Sulfasuxidine is the best of the poorly absorbed sulfonamides now available. It is effective against the *Shigella* group, and it has the advantage of an extremely low rate of complications. This drug to be effective must be used in large doses approximately five times those given above for the absorbed sulfonamides.

General measures used in treatment of any diarrhea include rest, a fasting period of 12 to 24 hours, and careful attention to the diet. Sedation is frequently useful when pain or vomiting is marked, or the patient is particularly restless. Cathartics should not be permitted.

The diet in general should be low in fat and roughage, high in protein. Continued

breast feeding is satisfactory when possible. The artificially fed babies should be given a dilute formula of skimmed or evaporated milk. The addition of protein hydrolysates or the use of one of the protein milks may be valuable in the early stages of treatment. The formula in these children may be made up with lactate-Ringer's solution as an aid in combatting acidosis. It is necessary when beginning feeding after the period of starvation to keep the feedings small and frequent, gradually increasing as the patient improves until the normal formula is resumed. The same is true with other children.

A number of special preparations, such as apple powders, foods combined with agar and kaolin mixtures have been used with varying degrees of success. They all seem to be helpful in the control of diarrhea.

SUMMARY

The management of the severe case of diarrhea requires a good laboratory for best results. The laboratory provides the means of intelligently combatting acidosis and dehydration, the usual cause of death when it occurs. Second, it provides a basis for intelligent use of specific therapeutic agents now available. With or without such facilities, however, the important thing to remember is that energetic treatment of the dehydration and acidosis does more to save life and shorten illness in young children than any other single measure. This combined with specific sulfonamide therapy will effectively bring about recovery of all but the most severe cases.

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CHRONIC COUGH IN CHILDREN*

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Like so many common symptoms, cough very often fails to fit into pathognomonic patterns, respond to recommended remedies, or receive the respect it deserves. Regardless of the cause, a persistent or severe cough can itself lead to destructive changes in respiratory surfaces, bronchial dilatation, and emphysema, as well as a number of other more remote but equally dangerous effects. With cough as a chief complaint, the list of possible diagnoses is long, and little point would be served by including it here; the more careful the clinical study, the longer the list becomes. Perhaps no common symptom is passed over lightly so often or treated so frequently without establishing a satisfactory etiology. It is as though physicians justifiably tire of continuously facing this diagnostic challenge and so take refuge in merely treating the symptom. Cough is essentially a protective mechanism. A physician many times does harm by injudiciously prescribing specific medication to suppress rather than aid the reflex.

Students, like their textbooks, place tuberculosis and bronchiectasis high on their differential diagnostic lists; commendable as the practice may be, we can certainly all agree that these are relatively rare causes of cough among children of the age when the complaint is commonest. Fortunately, these two very serious causes can be excluded readily by dependable objective means, but then there is a slump in diagnostic enthusiasm—what next? Forget about it and suppress the reflex? Tell anxious parents they need not worry? Depend on nature, time or a change in the weather?

*Read before the Sixty-sixth annual meeting of the Louisiana State Medical Society, Alexandria, May 6-8, 1946.

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Only very occasionally can the answers to these questions be "yes"; in most cases serious enough to be brought to medical attention, our work has only begun.

The pattern in the act of coughing is familiar: preliminary inspiration, closure of the glottis, increasing intrathoracic pressure, sudden opening of the glottis, and a strong expulsive effort. Whereas it certainly may be voluntary and though it may sometimes be withheld, it usually appears to be entirely reflex in nature. The younger the child, the more *inefficient* a cough appears to be. Expulsive efforts often seem purposeless, poorly directed, and ill-timed; tiring paroxysms are apt to follow apparently minor stimuli; major stimuli may be followed by only feeble, inefficient hacks. Aspiration of anything that happens to be present in the mouth, nose, or throat commonly occurs with the next inspiration. Stimulating impulses may arise from intrinsic or extrinsic irritation of the respiratory mucosal surfaces, excitation of nerve endings in the pleura, vagal afferents in other areas such as the esophagus, stomach, or external auditory canal (Arnold's nerve). Pressure adjacent to these structures may also produce cough. There are about seven other means by which irritants and secretions may be removed from the respiratory tract; the first three of these—ciliary action, bronchiolar peristalsis, and phagocytosis in the alveoli—might be termed "insensible" and apparently act adequately alone to remove normal secretions or small aspirated particles; cough probably occurs only when these three physiologic processes are overtaxed. The other four—postural drainage, hyperventilation, suction technics, and physical or chemical measures designed to reduce the viscosity of secretions or exudates—are ordinarily employed in turn only to aid the overtaxed cough.

Productive or not, cough of severe recurrent or paroxysmal nature in young patients often appears to require an inordinate amount of physical work causing all too apparent exhaustion. Obviously inadequate pulmonary ventilation, cyanosis, or convulsions are not unusual and may be accompanied by serious degrees of anoxia, respiratory acidosis, and petechial or gross hemorrhages in the brain or meninges; extensive encephalopathy has been ascribed to resultant circulatory changes.

In case histories, one is struck with the large number of potential inciting factors that may be elicited; it is usually difficult to ascribe chronic cough in a child to any single one. In many cases, familial allergic features (real or imagined) justify suspicion of atopy as a major or contributing cause. Tuberculophobia, which so often brings many of the milder cases to medical attention, is usually based on previous unfortunate familial occurrence; parents who have had experience with other serious or chronic pulmonary disease may be similarly apprehensive. A cough which is continuous, increasingly severe and productive, and aggravated by change in position, is particularly ominous; beginning in infancy and accompanied by recurrent diarrhea and wasting, there is legitimate reason to suspect fibrocystic disease with its familiar sequential pattern. A cough which follows aspiration of a foreign body, any operation on the upper respiratory tract, anesthesia, or inhalation of common irritants such as oils, kerosene, or powders, must always be regarded seriously.

Histories are usually replete with distinct evidences of chronic or recurrent infections and obstruction in the upper respiratory tract; these precede or accompany exacerbations of cough and fever of

variable degree and duration. These episodes are often justifiably diagnosed as bronchitis or pneumonia, initiated by fresh "colds" or any other common infection. There are many corroborative complaints attesting to the chronicity or frequency of these: poor appetite and weight gain, nervousness, fatiguability, and behavior problems of seemingly endless variety.

From careful description of a cough, the clinician often gains diagnostic information of localizing value, even though inaccurate accounts by distraught parents may require meticulous interpretation and evaluation. There is no doubt that a careful history — neglected as it has been in these past few busy years—will almost always furnish more pertinent clues to correct diagnosis than any other portion of the clinical study.

Though physical examination may yield surprisingly negative results, it more often discloses many abnormalities related as cause or effect to the major complaint. Signs of chronic infection are common, though variable in degree: faulty posture, small stature, anemia, poor muscle tone, weight loss, or inadequate gain. There is usually unmistakable evidence of disease about the pharynx, nasopharynx, nose, paranasal sinuses, ears, or regional cervical nodes. Visible, audible, and palpable obstruction to the nasal airway may exist. In many of these patients, characteristic pale, boggy, glistening nasal mucosae lend support to suspicions aroused by an allergic history. Such evidence, even when accompanied by distinct infection, is best documented by careful nasal smears prepared with Giemsa's or Wright's stain; nasal eosinophilia can be demonstrated frequently in these cases but is rare in nonallergic individuals. By simple testing, obstructive deafness can often be demonstrated; here otoscopic examination shows some retraction of tympanic membranes or signs of middle ear infection.

Conventional examination of the pulmonary fields is usually disappointing. Although respiration may be slightly increased, percussion note a bit hyperresonant, and breath sounds somewhat harsh, wide physiologic variations make these difficult to appraise. Scattered rales may be heard about the pulmonary hilar areas or bases but usually clear by the time of day these children are brought to the doctor's office; during exacerbations demanding home visits, indications of pulmonary disease are usually clearcut and multiple.

Whereas roentgenographic examination is a most valuable adjunct in studying these cases, it is often improperly employed as a shortcut to supplant equally dependable but more time-consuming diagnostic measures. Interpretations should always be correlated with other clinical findings, rarely trusted alone. To reenforce careful examinations, views of the chest and paranasal sinuses are taken almost routinely in any complete study of chronic cough in a child; lateral views of the nasopharynx are also occasionally useful. "Surprises" should always lead us back to the patient to recheck our observations and technics of physical examination but never to depend abjectly on the x-ray.

Appropriate serial tuberculin tests should be performed in all children, regardless of whether or not cough is present. Positive reactors may be disclosed more frequently among children with chronic cough; then the real problem is to differentiate between active or quiescent, infectious or noninfectious, and primary or reinfection forms of tuberculosis. Any serious infection responsible for chronic cough will generally be accompanied by measurable evidences such as fever, increased sedimentation rate, leukocytosis, and polynucleosis. If the cough is productive, proper examination of the sputum may directly and correctly define its cause. From infants and young children sputum is best obtained by nasopharyngeal swab or esophageal aspiration. In addition to the usual obvious indications for such study, occasional examples of mycotic infection can be discovered by this means.

Whenever there is reasonable evidence of pulmonary suppuration, complete broncho-

graphic study after instillation of lipiodol should follow as a matter of routine; only by this means can the presence of bronchiectasis be established with certainty.

When chronic cough is accompanied by symptoms and signs suggestive of the celiac syndrome, fibrocystic disease must be considered and pancreatic enzymes in duodenal contents investigated. Indeed, in some examples of this condition, symptoms of intestinal disorder are overshadowed by serious pulmonary manifestations; the presence of inspissated secretions accompanied by dilatation, atrophy and fibrosis of glandular structures all over the body furthermore suggests that such changes in pancreatic acini are only incidental to a generalized disease not yet completely understood. Examination of the feces should be a routine part of any investigation to determine the cause of chronic cough; common parasitic diseases (ascariasis, strongyloidiasis, and hookworm infection), in which the responsible organism ruptures the pulmonary capillaries and alveoli, may be recognized. These undoubtedly produce specific respiratory symptomatology as well as constitutional effects.

After analysis and correlation of thorough diagnostic procedures, specific therapy may be selected. While such studies are in progress, however, diligent efforts should always be made to treat contributory or associated infections, relieve obstructions, aid in removal of secretions, and improve nutritional deficits. The simplest technics are still most generally effective: intelligent administration of nose drops, postural drainage, hyperventilating exercises, and increased humidity.

In children, use of any of the common agents for shrinking nasal mucosae should be interrupted for about twelve hours every few days to minimize undesirable secondary irritative effects. The dropper is preferable to the spray, and accumulated secretions should of course be removed before drops are instilled. The simplest and generally most efficient technic (Parkinson) employs the lateral head-low posture. There is little or no justification for including various sulfonamides in nose drops or sprays. Aerosol solutions of penicillin or streptomycin are certainly beneficial in cooperative selected patients. Most infants and young children prefer to sleep in the prone or even knee-chest position, so effective for clearing secretions from the large respiratory passages; even better drainage can often be secured by elevating the foot of the bed a few degrees. With a little ingenuity, simple hyperventilating exercises attractive to the child can usually be devised, ranging all the way from balloon-blowing to strenuous trapeze exercises.

Increased humidity—obtained by a number of modifications of the old-fashioned familiar croup tent — is helpful often enough to deserve trial along with these other simple procedures. Care should be taken to avoid drafts or undue exposure when steam is being used.

The common "tonics" ordinarily employed are rarely effective in the presence of chronic infections. The rather severe hypochromic anemias seen so frequently can be blamed about equally on infection and deficiency and are best treated directly by transfusion. Various hematinics and vitamin mixtures have a valuable place after such preliminary correction, and their use is certainly to be encouraged in the usual prolonged convalescence.

Unless specific local indications exist, adenoidectomy or tonsillectomy has little to offer the child with chronic cough. Certainly when there are allergic manifestations, this operation should always be delayed until the effect of environmental changes, removal of offending substances, or desensitization has been observed.

With or without demonstratoin of specific sensitivities, the child whose chronic cough appears in any way related to nocturnal environmental influences deserves the opportunity of sleeping in a room from which all possible offenders have been removed. When correlation of positive skin tests and symptoms can be demonstrated, elimination of allergens is generally preferable to desensitization in the young child; unfortunately, the former may be difficult

or impossible. The effect of newer antihistamine preparations has not been critically analyzed in respiratory allergic disorders of children, but is certainly promising enough to deserve trial.

When a cough is severe and obviously not accomplishing any desirable function, when its cause has been removed but it persists as an irritative or "habit" mechanism, or when it disturbs necessary periods of rest, adequate sedation may be advisable. Phenobarbital is still the most useful sedative in pediatric practice. Belladonna or opiates should rarely be used, as they increase the viscosity of secretions; when sputum is scanty or absent, however, small doses of codeine may be indicated.

An adequate fluid intake, combined with increased humidity, liquefies sputum more efficiently than the best expectorant drugs: ammonium chloride, ipecac, and potassium iodide. These are all difficult to disguise, and are frequently refused by young patients who appear to need them most. Every physician has his own favorite prescriptions; I know of none that is always successful.

No attempt has been made in this paper to review all the causes, diagnostic studies, and therapeutic measures for chronic cough in children. Such a work would fill several volumes. This common pediatric problem illustrates beautifully the need for seeking and removing the cause while treating the symptom. Perhaps in academic circles we seek too much and treat too little, while in practice the converse criticism often applies.

DISCUSSION

Question: What is the treatment of cystic disease of the lung, especially a large cyst of one lung.

Answer: Such cases are unusual. Treatment consists essentially of surgical removal at the earliest possible date. Once the inevitable infection has occurred, therapy is more difficult and hazardous.

Question: Remark on the use of CO_2 to liquefy secretions and aid cough.

Answer: I suppose you mean 5 per cent CO_2 in oxygen; this may be difficult to use adequately in small children, and I have no conviction that it is more valuable in the therapy of cough than the common alkaline cough mixtures.

Question: In chronic cough of undetermined cause, do you think that the bacterial vaccines on the market are of some benefit?

Answer: Certainly the literature is confusing on that point. From personal experience, I have yet to see such a case I believe has been benefitted by the use of commercial bacterial vaccine. Then too, it is difficult to divorce the effect of such vaccines from any other therapy the physician has employed. From an immunologic point of view, we have little reason to expect results from vaccine.

You are perhaps all familiar with the work of Diehl and his associates—for a large group of University students, the use of oral or injectable "cold vaccines" accomplished nothing in a well controlled evaluation of these products.

MENINGOCOCCAL ADENITIS

CASE REPORT WITH DISCUSSION

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Chronic localized complications of meningococcal infection outside of the nervous system are not uncommon. Arthritis[12] has been encountered perhaps most frequently, with reports giving figures varying from 4.6 and 20 per cent as the proportion following meningococcal infections of all types. Endocarditis,[9] conjunctivitis,[11] panophthalmitis,[5] urethritis,[4] epididymitis,[1, 8] salpingitis,[8] parotitis,[5] purulent pericarditis[5] and nephritis[1] have all been reported as complications of meningococcemia. From all of the instances cited, Neisseria intracellularis has been successfully isolated.

The case here described is, to our knowledge, the first report of suppurative adenitis due to the meningococcus and presumably secondary to chronic meningococcemia.

CASE REPORT

An eleven year old white school girl, E. H., was admitted to Charity Hospital November 6, 1945. The chief complaint was fever, which had persisted for two weeks.

Onset and Course: On October 24, 1945, two weeks before admission, the patient returned from

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school with pain in the right popliteal space. That night she had fever of 102° (orally) but the pain disappeared. Fever continued during the next two weeks, however, and on November 5 pain developed in her right elbow. Her temperature rose to 105°, and she vomited several times. All pain had disappeared by the next morning but the parents brought the child to the hospital. There had been no swelling of the joints nor any other abnormal manifestations.

The family consisted of the father, mother and seven siblings all of whom were well. The child's past history was non-contributory.

Physical Examination on Admission: Temperature 101.4°, pulse 105, respirations 28, weight 60½ pounds. Examination revealed a well nourished, well developed white girl, slightly pale, but not acutely ill. Pertinent findings were: (1) Pharynx and tonsils moderately injected; (2) small, firm, discrete, non-tender nodes present in the cervical, inguinal and axillary regions; (3) a soft blowing systolic murmur heard at the apex, not transmitted.

Laboratory Data on Admission: Hemoglobin 12 grams, red blood cells 4,100,000, white blood cells 12,850, polys 78%, lymphocytes 18%, eosinophiles 3%, monocytes 1%. Sedimentation rate 41 mm/hr., corrected (Wintrobe). Urinalysis negative, serology negative, Mantoux (1-10,000) negative at 24 and 48 hours. Blood culture negative, throat culture negative, stool culture negative. Blood agglutinations: Twice negative for *E. typhi, paratyphi B., B. abortus* and Proteus X19.

Clinical Course: The impression on admission was "possible rheumatic fever" and treatment was symptomatic.

Throughout her hospitalization the patient had no complaints and did not appear acutely ill. The fever spiked to 105° the first two days and to 103° on the third day. It then hovered around 100° until the eighth day when it reached 103°. On the ninth the highest point was 104° and on the tenth and eleventh, 102°. Thereafter there was gradual decline to normal by the end of the third week, continuing so until discharge ten days later.

The systolic murmur became progressively louder but did not change in character. On November 18, the thirteenth hospital day, the spleen was palpated 1 cm. below the costal margin. It was firm and not tender. On November 26, the twenty-first hospital day, an erythematous, macular rash appeared on the abdomen, chest and back. This lasted for one day and was asymptomatic.

Laboratory Work During Hospitalization: Electrocardiograms taken on November 7, and November 16 were within normal limits. Repeated blood studies revealed no change except for a slowly decreasing sedimentation rate to 7 mm/hour on December 1. Repeated urinalyses showed no abnormalities. Blood cultures taken on November 9 and on November 16 remained negative. Brucellergen intradermally yielded no reaction. Urine culture and repeated stool cultures showed no pathogenic organisms.

The patient was discharged on December 5, 1945, after four and a half weeks in the hospital without further clue as to diagnosis.

Interval at Home: She was seen in the outpatient department on December 20, 1945, two weeks after discharge, at which time her temperature was 100°. She was said to have been having low fever every afternoon for one week. She had also had a macular rash on trunk and thighs for the three days before re-admission. This rash apparently was similar to that which she had had on the ward. The patient was sent home for Christmas and was next seen on December 27, when she was re-admitted to the hospital.

Second Admission: Afternoon elevation of temperature had continued during the week before re-admission and six days before admission a slightly painful lump had appeared in the left groin. On examination the temperature was 99.4° and a soft apical systolic murmur was present. There was a slightly tender mass, two inches by four inches, in the left inguinal region, which was not fluctuant, red or hot. It was thought to be a lymph node. No vaginal pathology or leg lesion was present and no other glands had changed.

The blood picture was essentially the same except that the sedimentation rate was now 14 mm/hour, corrected. The urine, Mantoux (1-100), Frei test, and electrocardiogram were negative.

Course in Hospital: During the next two weeks the temperature was not elevated but the mass persisted. The lymph node was thought to be softening and on January 7, 1946, ten days after admission, sulfadiazine, 1 gm. t.i.d., was started. The gland was fluctuant on January 10, 1946, three days later, and was aspirated, yielding greenish pus. Small bodies suggestive of gram negative diplococci were observed in direct smears of this material.

While waiting for growth of cultures which had been made from the aspirated pus, sulfadiazine was continued at the same dosage. On January 13, however, three days after aspiration and six days after starting sulfadiazine therapy, the temperature went up for the first time to 102°. The next day it reached 104.4°. On the following day the abscess was incised and drained, yielding thick green pus. In contrast to the first specimen, no organisms were seen on direct smear. The dosage of sulfadiazine was increased to 1 gm. every four hours, but during the next two days the temperature again reached 104.4° and a fine macular erythematous rash appeared. A blood count showed 4,000 white blood cells per cu. mm. and it was thought the patient was having a drug sensitivity reaction. Sulfadiazine was therefore discontinued and penicillin 20,000 units was given

every three hours for six days. The temperature gradually returned to normal after four days of this therapy. The wound healed steadily and was closed in about two weeks.

After three weeks of normal temperature it was decided to test the validity of the assumption that the febrile episode was due to sulfadiazine sensitivity. A single dose of 1 gm. of sulfadiazine with 2 gms. of sodium bicarbonate was followed within two hours by nausea, vomiting, malaise and a fever of 102.8° which returned to normal over the next 36 hours. No rash was noted.

No further untoward event took place and the child was discharged on February 15, 1946, after a hospital stay of seven weeks.

She has been followed in the out-patient department since discharge and has continued in good health with no recurrence of symptoms.

The final diagnosis was: (1) Suppurative lymphadenitis, meningococcal; (2) chronic meningococcemia; (3) sulfadiazine sensitivity.

Bacteriology: Because of the observation of gram negative diplococci in the direct smear of aspirated pus, special cultural procedures were undertaken. The aspirated material itself was incubated and the usual media used in this laboratory, infusion broth, infusion agar and infusion agar with 1 per cent human blood, were inoculated. In addition, certain special procedures were followed in an effort to isolate anaerobic bacteria. Brain-heart infusion broth and Brewer's Thioglycollate Broth (Difco) were inoculated and kept in the standard incubator. These media have, on many occasions, grown anaerobic or partially anaerobic bacteria successfully. Finally, chocolate agar plates were seeded and placed under increased CO_2 tension.

It was only from the chocolate agar—increased CO_2 procedure that growth was obtained. From material aspirated on January 10, 1946 and the pus from incision and drainage on January 15, very tiny, smooth translucent colonies were obtained. Organisms from these colonies were typical gram negative biscuit shaped diplococci. The oxidase reaction was positive. Fermentation reactions showed acid production with both maltose and glucose.

A culture was sent to the National Institute of Health for further identification and agglutinations with type specific sera and was reported as meningococcus, Type I.

DISCUSSION

In view of the absence of any positive bacteriologic data on the patient's first admission one can only speculate on the course of events. With no findings referable to the central nervous system and no history of a rash, meningococcemia was not suspected. The rash which was reported on the twenty-first hospital day of the first admission occurred after the temperature was normal, was only transitory, and was certainly not typical of the rash of meningococcemia. The same remarks apply to the rash reported during the interval between the two hospital admissions.

In retrospect, increased importance might have been attached to the history of joint involvement. Such complaints of fleeting pain are not infrequently seen, however, as a non-specific phenomenon in infectious diseases. The various reports of joint involvement in meningococcemia stress the point that there is an "arthralgic" type, characterized chiefly by pain and some swelling but without evidence of suppuration. If, as seems likely, the original complaint, in October, 1945, marked the onset of meningococcemia, one would classify the joint involvement as of the arthralgic type.

Apparently there were no other localizations, and the absence of growth in any of the four blood cultures taken at the first admission indicates that such bacteremia as was present was probably not heavy. It is possible that had the blood cultures been made with special technics later used to recover the organism from the abscessed inguinal gland, positive results might have been obtained. Yet, there appeared to be no reasons clinically at the first admission, to suspect an organism which required special conditions for growth. It has been advocated by some that in any condition where an infection is suspected and no organisms recovered by usual methods, whether it be from blood or pus, the laboratory should employ special anaerobic technics routinely. Such a procedure would be of help in many cases but is obviously expensive and would require more laboratory staff.

The interval from apparent onset of the disease, October 24, 1945 to the time the gland was first noticed, December 21, 1945, was almost two months. Furthermore, she was afebrile during the last ten days of her first hospital stay and during her second admission until after the gland had become fluctuant. During the interim she was reported to have had only a slight afternoon

rise in temperature. One can postulate either a continuing meningococcemia of very low degree unaccompanied by significant fever and followed by late localization in the inguinal gland, or, perhaps a seeding of the organisms in the particular gland during the first febrile episode, with a long period of incubation before suppuration took place. Neither explanation is completely satisfactory.

No chemotherapy was given at the first admission and sulfadiazine given on the second admission served only to produce a sensitivity reaction. Since at the time the penicillin was started, the gland had already been opened and was healing, it is doubtful if penicillin played an important role in the child's recovery. It is interesting and important to note, in days when chemotherapy is employed on slightest provocation, that the child's own defense aided by mechanical drainage of an obvious suppurative process, successfully cured the disease.

SUMMARY

A case of isolated suppurative lymphadenitis is reported. The etiologic agent was a meningococcus, Type I. It is believed likely that the lesion was a complication of chronic meningococcemia although positive blood cultures were not obtained.

We wish to thank Dr. Marion Hood of the Bureau of Laboratories, Florida State Department of Health, formerly of the Department of Public Health, Louisiana State University, School of Medicine, for her interest in this case and for her help in writing this report.

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THE WELL CHILD*

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The "well child", at first glance, would seem to be almost too simple a subject for discussion, but more careful perusal reveals its complexities. Our children are the nation's collateral for long term investments. Aside from the purely practical business of admitting that an ounce of prevention is worth a pound of cure, we all agree it is the aim of a democratic society to see that the sick infant or child becomes well and that the healthy infant or child stays that way. Our aim is not to provide husky atomic bomb fodder at some future date, but rather to provide an even start when the children of today have developed to the point they can enter the competitive race of tomorrow.

Synonymous with the "well child" is the "happy child." Certainly no child is completely well who, though sound of body, has a sense of insecurity in his environment. It is the purpose of this paper to review briefly some of the salient principles which foster optimal physical as well as mental growth and development.

PHYSICAL GROWTH AND DEVELOPMENT

The strides made by the biologic sciences within the past few decades have greatly lengthened the average life span. A considerable part of this increase is due to our ability to carry children through the hazards of childhood. Pasteurization of milk was a forward step in this program. Development of scientific infant feeding reduced tremendously the dreaded scourge of cholera infantum, or infantile diarrhea. Reduction in incidence of the contagious childhood diseases, by extension of our prophylactic immunization programs, is

*Condensation from an address delivered at the Health Education Workshop, Southeastern Louisiana College, Hammond, La., on September 2, 1946.

common knowledge. Unfortunately, however, there are still some serious childhood diseases for which no protective vaccines have been developed. We are concerned about the recent outbreak of poliomyelitis, yet we know practically nothing about its transmission. Rheumatic fever is another childhood disease of which our knowledge is limited. It is even more crippling than poliomyelitis, although its effects are not as obvious. We can all expect that continued research in these fields will eventually bear fruit. Particularly encouraging are such developments as the adaptation of various strains of poliomyelitis virus to inexpensive laboratory animals, such as cotton rats and white mice. Probably such adaptations will stimulate vital research in this field and will eventually amplify our knowledge of the epidemiology of this disease. Many reports of chemoprophylaxis for group A streptococcal infections have been favorable; these measures may prove to be potent weapons in the control of rheumatic recrudescences.

Many parents still do not appreciate the practice or wisdom of visiting a physician primarily to keep their child well. An annual physical examination should be urged for every apparently healthy school child. During this visit there should be a comprehensive review of the past medical history, a complete physical examination, such labatory work as may seem indicated, and a frank discussion of all the child's physical and mental problems. It is not difficult to recognize the healthy child. He is alert and active, with clear skin and bright eyes. His tissue turgor is good, his muscles firm, and his body strong. During play periods he participates eagerly and vigorously. He gains continually in size and weight throughout childhood, although growth increases are not constant; there are periods of relatively rapid and slow growth increases, particularly in the preschool period. Isolated comparisons of a child's height and weight with many of the standard charts may have little significance, since some children are inherently larger or smaller than the average. Evidence of a constant falling off or gain from standard curves, as determined by serial "longitudinal" measurements, are much more indicative of abnormality.[1]

The gospel of clean milk and water supplies should be reiterated to lower still further the incidence of typhoid and undulant fever, streptococcal sore throat, dysentery, and other less common milk and waterborne diseases. An extensive educational program has greatly reduced tuberculosis in this country; childhood tuberculosis can be practically eliminated by periodic examination of adults with whom children are in close contact. This is desirable because children are most susceptible and least able to overcome this infection. Intestinal parasitism in children is still a major problem in this region. By education in proper hygienic methods and by careful segregation and treatment of infected individuals these debilitating conditions would assume a position of minor import.

Through school health programs, immunization procedures can be publicized. Every child of school age should have been successfully vaccinated against smallpox and satisfactorily immunized against diphtheria, preferably in early infancy, with supplementary immunization at school entrance. All infants should be immunized with pertussis vaccine and routine mimunization of children with tetanus toxoid is now recommended. There is considerable evidence that many of the multiple antigens (such as combined diphtheria-tetanus toxoid) are satisfactory immunizing agents. Where water supplies are doubtful, typhoid vaccination should be mandatory. It has been my practice to administer a "booster" dose of 1.0 c.c. of diphtheria-tetanus toxoid one year after initial immunization. In addition I apply tuberculin patch tests (until positive) and Schick tests at regular intervals. A suggested immunization plan which is in accordance with the recommendations of the Committee on Therapeutic Procedures of the American Academy of Pediatrics[2] is presented in table 1.

In the field of nutrition the physician should play a leading role. Many parents

TABLE 1
SUGGESTED IMMUNIZATION PROGRAM FOR THE WELL CHILD

Age	Procedure
3 months	Smallpox vaccination — primary reaction
6 months	Pertussis vaccination (with Phase I vaccine) 3 injections
9 months	Combined diphtheria-tetanus immunization 2 injections
18 months	Schick test
2 years	Tetanus toxoid (booster — 0.5 c.c)
3 years	Tuberculin test
6 years	Smallpox vaccination — primary, secondary, or immune reaction Schick test Tuberculin test
9 years	Tuberculin test
12 years	Smallpox vaccination — primary, secondary, or immune reaction Schick test Tuberculin test

have a surprising lack of knowledge as to just what constitutes a child's dietary needs and common advice to "eat a good diet" is therefore often meaningless. Almost daily the pediatrician is consulted by parents who feel that their children need some tonic or vitamin. In most instances the only necessary therapy is an adequate well-balanced diet. In addition the physician can alleviate the anxiety of many parents by simple explanations of the variation in growth and metabolic needs that occur throughout childhood. Stress should be laid on the expected reduction in appetite during the preschool period when weight increases are at a minimum. We are a nation of faddists, and at present vitamins are fashionable. One might say that we are being "vitaminized to death." If we are to believe the advertisements, there is practically nothing that vitamins will not cure. Yet the simple truth of the matter is that no vitamin deficiences will develop in normal children on well-selected, adequate diets.

MENTAL GROWTH AND DEVELOPMENT

Pediatrics was one of the first medical specialties to recognize the importance of preventive medicine. Every child should have a program of medical health supervision and preventive mental hygiene adapted to his individual needs. Although final care rests with the parents, the physician, psychiatrist, and educator must all cooperate and offer them leadership.

Just as the nuclear physicists have far outdistanced the political scientists, so in pediatrics have the objective sciences surpassed subjective ones. The child guidance movement is a mere infant among older branches of pediatrics; one naturally expects such a baby to make errors before it grows to maturity. Handicapped by a cumbersome terminology and overburdened by the necessity of treating physically ill children, physicians initially displayed a good deal of inertia toward mental hygiene. Unduly influenced by the behaviorists, the child guidance movement was ultra-scientific in the early stages. Fortunately, the pendulum is now swinging back to a more common sense, middle-of-the-road position encouraged by the active cooperation of physicians who deal with children.

Not long ago infantile debility, or marasmus, was one of the serious common diseases of infancy. Frequently babies living in hygienic surroundings but without benefit of devotion became marasmic, whereas infants living in poorer homes but with attentive mothers often overcame the handicap of their environment and developed normally.[3] It was the need for affection with its subsequent feeling of security that made the difference. As a result the system of foster home placement rather than institutional care has been developed for infants who have no adequate person to care for them. Hospitals are beginning to recognize this necessity for fondling; several have installed old-fashioned rocking chairs in their infant wards; nurses are instructed to rock infants for periods each day. It is as important to gratify the emotional needs of infants as it is to administer diphtheria toxoid. The latter may prevent disease, whereas the former may avert subsequent equally disturbing behavior problems.

An excellent example of carrying the scientific method beyond the range of sanity is illustrated by some fortunately obsolete technics in the feeding of infants. Babies are more old-fashioned than the "surrey with the fringe on top." A newborn infant of 200 years ago could not be differentiated from a newborn of today—both are babies. As Aldrich[4] has pointed out, the developing

child is under the compulsion of two forces; preordained growth drives (physical, emotional, and mental) and the opposing impact of the customs of society. The latter force may arbitrarily dictate to a child how much and when to eat, sleep, or even play. Contributions medical science has made to lowering infant mortality have also inadvertently tended to dogmatize infant feeding in the direction of rigid routines. Such systems have completely neglected emotional needs and individuality of infants, even implying that all babies are born with a "gilt-edged seal" that they are "three or four hour interval feeding babies." Fortunately, most (but not all) infants are able to adjust themselves to being fed "by the hands of the clock." We now realize that "infants are people", each with his or her own inherent characteristics. It has been demonstrated that every baby has certain rhythmic time intervals between hunger pangs. It would seem logical, therefore, to offer feedings at such physiologically rhythmic times, since during the newborn period nursing is obviously one of his greatest satisfactions. An excellent illustration that infants and children have considerable capacity for self-regulation is the oft-cited experiment of Davis[5] on the self selection of food. Fifteen infants of weaning age were allowed to choose what they ate from a variety of natural simple foods essential to health and growth; no comments or suggestions were offered by the attendants. The children rarely mixed their foods, they even ate salt separately. Some of the meals were strange affairs consisting of as high as four or five bananas. One child was reported to eat 10 eggs at a single sitting. Yet the nutritional results over a long period of time (involving some 36,000 meals) were excellent. The group equalled or surpassed conventional standards of growth, were free of digestive upsets, and presented no eating problems. An analysis of each child's food intake over a long period showed that they all were within the limits of scientific nutritional standards. These experiments did not prove that a child can be depended upon to select a proper diet under ordinary circumstances, but only when conditions are favorably controlled, that is, children without preformed likes and dislikes, exposed to natural, unprocessed foods only).

Feeding, however, is only one of many problems in which the inherent characteristics of the child must be adapted to the mores of society. In order to become an acceptable member of society the child must eventually be trained for bowel and bladder control. He must learn to suppress some of his possessive, selfish animal instincts and make his contribution willingly to the group. The negativistic period of childhood should be passed without disrupting the rest of the household. Normal sex drives of adolescence must be kept within the structure of our moral code.

It is the duty of all of us to help each child make these adjustments as naturally as possible and without imposing strict regimentation upon him. I believe this can best be done if we remember that children are all individuals and cannot be compressed into a single mold. In an atomic age, Brennemann's statement[6] seems particularly apt: "The child who presents no conflicts and no deviations from a conventionally ideal behavior is a dud; he will never make a noise in the world."

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SOME CORRECTIBLE ANOMALIES OF THE HEART AND GREAT VESSELS

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With recent advances in thoracic surgery interest in congenital heart disease has shifted from the experimental laboratory to daily medical practice. The purpose of this

paper is to review and summarize the background of this subject, to discuss recent clinical advances in diagnosis and treatment of certain congenital lesions, and to summarize results of their surgical treatment.

ANIMAL EXPERIMENTAL BACKGROUND

No attempt will be made to cover this field entirely, but a few interesting papers will be cited. In 1909, Bernheim[1] produced mitral stenosis in an animal, studied resultant changes in circulation, and attempted to determine if correction of the stenosis would restore normalcy. Cushing and Branch[2] probably first successfully used a direct approach to the valve through an incision in the cardiac wall of an animal.

In 1910 Alexis Carrel[3] reported his famous studies on experimental methods of diverting blood during operations on the thoracic aorta by: (1) placing a paraffin tube into the aorta, and (2) lateral diversion through a paraffin tube extending from an incision in the apex of the left ventricle to the descending aorta. Included with the above were descriptions of successful suture of the ascending aorta, end-to-end anastomoses of the descending aorta, and successful graft of a segment of vein preserved in cold storage to an artificially produced defect in the descending aorta. Four years later he described the use of a soft-jawed mass pedicle clamp with which all blood flow to and from the heart could be halted for 2½ to 3 minutes without permanent damage.[4] Employing this instrument he was able to perform a number of experimental operations and to demonstrate a wide range of feasible procedures.

In 1922 Allen and Graham[5] described a cardioscope with an attached knife which could be inserted through a small incision in the myocardium. With later improvements in technic, Allen[6] was able to insert this cardioscope through the left auricular appendage and change mitral stenosis to insufficiency with resultant improvement in pulmonary circulation.

There was a surprising lapse of interest in experimental cardiac surgery for about 20 years. In 1943 Shaw and co-workers[7] used sling sutures about the vena cavae and azygos vein to interrupt blood flow permitting opening of the myocardium and visualization of the valves; he found that operations on valves of the right side of the heart were accomplished with greater ease than those on the left. Harken and Glidden[8, 9] discussed various cardioscopes and described a new instrument providing better visualization than older types.

In 1942 Mandelowitz and Leslie[10] anastomosed the pulmonary artery to the left auricle with results imitating those seen in circulatory derangements typified by the tetralogy of Fallot. Relative and absolute increases in systemic over pulmonary blood flow were observed, but anoxemia occurred only after exercise. Of four dogs surviving anastomosis, only one developed osteo-arthropathy; this animal had an absolute increase in systemic volume and lived long enough for the arthropathy to develop. Since there was no anoxemia in this dog the authors attributed these peripheral changes to increased periosteal nutrition and proliferation.

To maintain life during diminished or arrested pulmonary circulation, Gibbon[11] used a DeBakey pump[12] to withdraw blood from the jugular vein, which was then oxygenated and returned to the aorta via the carotid artery. With this procedure three animals survived complete pulmonary arterial occlusion of 12-18 minutes' duration for 9-12 months.

CLINICAL DIAGNOSTIC AIDS

To familiarize diagnostic procedures Robb and Steinberg[13] in 1939 added the use of a contrast technic employing diodrast given intravenously to permit more accurate roentgenologic study of various circulatory anomalies. Reyes and his associates[14] improved and modified Robb and Steinberg's technic by adapting apparatus to secure simultaneous views of the heart from several angles. They reported that normal and pathologic images could be easily differentiated and described the typical changes in orificial and infundibular types of pulmonary stenosis, patent interventricular septum, transposition of the great vessels, tetralogy of Fallot, and interauricular

septal defects. They proposed a special method for demonstrating patent ductus arteriosus: the radiopaque material was injected through a thin trocar in the left brachial artery and passed by retrograde flow into the aortic arch, through the patent ductus arteriosus and into the pulmonary artery.

Stewart and associates[15, 16] used a motion picture camera to follow the course of radiopaque material fluoroscopically visualized.

Increasing experience with such means for demonstrating blood flow should facilitate more accurate anatomic diagnosis and selection of cases suitable for surgical correction.

CONGENITAL DEFECTS AND ATTEMPTS AT SURGICAL CORRECTION

Before entering into a discussion of this phase of the subject, credit should be given to Maude Abbott,[17] whose monograph reviewing and classifying 1000 examples of congenital heart disease did so much to bring order from chaos. Maliner and Borow's[18] clinical modification is, however, more practical, classifying most common types into those with and without cyanosis, realizing that some of the latter under severe stress or strain may temporarily show cyanosis. The most common of the acyanotic group are: (1) interventricular spetal defects of smaller size, (2) patent ductus arteriosus, (3) foramen ovale patency, (4) coarctation of the aorta, (5) subaortic stenosis (very rare). The cyanotic group exhibits such basic lesion as (1) pulmonary stenosis (especially with the tetralogy of Fallot); (2) large septal defects (cor bi- or tri-loculare); (3) transposition of the great vessels.

PATENT DUCTUS ARTERIOSUS

Since this subject has been covered by very competent writers, little space will be devoted to it here. In 1940 Gross[19] became the first in this country to close a patent ductus successfully and much credit is due this noted surgeon for his stimulation of interest in this and related problems.

Burch[20] reviewed practically all of the available case reports prior to March, 1944, and found that the average surgical mortality was 8.5 per cent in uninfected ducti, the rate varying, however, with the surgeon and his experience. He recommended the procedure for all patients with this anomaly and superimposed endarteritis, since more than 50 per cent of such patients have been cured by ligation, whereas this condition was previously hopeless. Touroff[21] also strongly recommended ligation when endarteritis is present. Since the publication of these two papers, however, Dawson and Hunter[22, 23] have reported spectacular results in the cure of this conditin with massive doses of penicillin, when the infecting organism is sensitive to this drug.

Thus it seems likely that at present there are four indications for ligation of the patent ductus: (1) progressive cardiac failure; (2) discernible impairment of mental and physical growth; (3) high pulse pressure and, (4) endarteritis when the infecting organism is resistant to available chemo-therapeusis.

Gross[24] has advocated complete surgical division, when possible, as more satisfactory than simple ligation or ligation and wrapping with cellophane to stimulate fibrosis.

PULMONARY STENOSIS

The heading pulmonary stenosis here is misleading in that it is merely the basic lesion present in a group of defects which have, largely through the work of Blalock and Taussig[25] become amenable to surgical treatment. These authors, quoting Lundsgaard and Van Slyke,[26] cite four factors in the production of cyanosis: (1) height of hemoglobin; (2) volume of venous blood shunted into systemic circulation; (3) rate of utilization of oxygen by peripheral tissues, and (4) extent of aeration of blood in the lungs (most important).

After producing pulmonary stenosis in dogs, the idea of anastomosing a systemic artery to one of the pulmonary arteries in order to increase pulmonary circulation was conceived. Following these experiments, three human patients were operated upon, using in the first case an anastomosis between the end of the left subclavian artery

to the side of the left pulmonary artery; in the second case (in which there was a right aortic arch) the end of the innominate to the side of the left pulmonary, and in the third the end of the innominate to the side of the right pulmonary. Ether and oxygen anesthetic was used in the first case, intratracheal cyclopropane in the second and third. Although no marked impairment of systemic circulation to parts deprived of their major arterial pathway was noted, simultaneous dorsal sympathectomy was suggested to minimize this hazard. Following successful transplantations there has been: (1) a pronounced decrease in cyanosis and dyspnea with an increase in exercise tolerance; (2) decrease in erythrocyte count, hemoglobin, and hematocrit; (3) a fall in oxygen capacity and increase in oxygen content of arterial blood, and (4) most importantly, a decided rise in oxygen saturation of arterial blood.

Experiments are continuing and a great deal of attention is being directed to the relative merits of end-to-end and end-to-side anastomoses. Results of side-to-side anastomosis of the aorta to the left pulmonary artery have been found discouraging. The suture method of anastomosis is preferable to Blakemore's vitallium technic[27] as the latter would not permit increase in caliber with growth.

Though the likelihood of thrombosis at the anastomotic site might theoretically be reduced by the use of anticoagulants, such as heparin or dicumarol, attendant risk of hemorrhage has made this issue controversial.

Anomalies in which such transplantations are indicated include those in which the primary difficulty is inadequacy of blood flow into the lungs, such as, tetralogy of Fallot, complete pulmonary arterial atresia, absent or defective right ventricle, truncus arteriosus with bronchial arteries, or even a single ventricle with a rudimentary outlet chamber in which the pulmonary artery is small. Before considering operation the clinician should be sure that there is: (1) roentgenographic evidence of a small pulmonary artery, and (2) clinical or roentgenographic evidence of the absence of pulmonary congestion. In differential diagnosis complete transposition of the great vessels, right aortic arch and the Eisenmenger complex must be considered. Some apparent failures after operation may be due to irreversible changes in the capillary bed of the lungs caused by prolonged deficiency of blood flow.

COARCTATION OF THE AORTA

Since we Americans are a patriotic group, we always prefer to attribute to one of our nationality the honor of originating ideas and methods. It seems, however, that Alex Carrel,[3] a Frenchman, performed the first animal experimental end-to-end suture of the aorta followed by recovery, and Crafoord,[28] a Swede, apparently first operated successfully on two cases of aortic coarctation. The latter clamped and resected the constricted segment and reanastomosed the ends.

Gross and Hufnagel[29] became quite interested in this lesion and reviewed the advantages and disadvantages of procedures such as those advocated by Blalock and Park[30] Carrel,[3] and Blakemore and Lord.[27] After animal experimentation Gross[29] treated two patients according to his own technic which closely resembled that of Crafoord.[28] In all four cases reported as successfully treated by these two surgeons, excellent results have been obtained.

TRACHEAL OBSTRUCTION FROM A VASCULAR RING

Again Gross[31] reported that vascular rings within the mediastinum are of two types: (1) the aortic arch absent from its normal position in front of the trachea, coursing instead behind the esophagus, the pulmonary artery lying in its normal position in front of the trachea but anchored firmly by a patent ductus arteriosus or remnant to the distal part of the aortic arch. Thus the trachea is compressed anteriorly by the pulmonary artery, and the esophagus is compressed posteriorly by the aortic arch. Division of the patent ductus arteriosus or its remnant should give relief. (2) The second type is a divided or split aortic arch, one limb passing posteriorly, then to the left behind the esophagus, and the other

coursing to the left in front of the trachea, the two limbs then converging to form the descending aorta. Either or both limbs may rise to major arteries, and the limbs may be of the same or unequal size. The tracheal and esophageal constriction may be relieved in some cases by dividing the ring, while in others this operation is impossible without severing vital arterial pathways.

Gross[31] reports a four month old infant with difficult wheezing respiration since birth, the tracheal obstruction resulting in four attacks of tracheitis of sufficient severity to require hospitalization. Roentgenographic evidence of a vascular ring was found and the suitable operation performed, both types of rings described above being found. The procedure was well tolerated by the patient with marked alleviation of symptoms.

Diagnosis is based, in this condition, upon the demonstration of indentation of the esophagus by barium study, and anterior constriction of the trachea shown by roentgenogram after lipiodol instillation with anteroposterior and lateral films. Only in certain cases does the presence of a vascular ring produce symptoms, but when symptoms are present; surgical correction is indicated.

SUMMARY

Accurate diagnosis followed by carefully planned surgical treatment is no longer an experimental procedure but rather a clinical reality for many types of congenital heart diseases. Animal experimentation relative to the subject is considered, together with recently described diagnostic aids and clinical reports of surgical treatment of certain anomalies.

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SPLENOMEGALY WITH CIRRHOSIS OF THE LIVER COMPLICATING JUVENILE DIABETES

A CASE REPORT WITH SPLENECTOMY

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The following case is reported because of its interesting features and because no similar case has been found in a search of the literature.

CASE REPORT

L. S. B., a white male, aged 13, was referred to me on November 22, 1943 because of generalized edema of the feet, scrotum, abdomen and face. He had been known to have diabetes for the previous two years and had been under medical treatment. His original complaint was polyuria and inability to gain in weight. He had been on a restricted diet but without special supervision. He had been accustomed to taking 10 units of protamine zinc insulin and 15 units of regular insulin mixed in the same syringe before breakfast. At noon he would take 10 units of regular insulin and 15 units of regular insulin before supper. For the previous week he had been troubled with diarrhea, having as many as 15 movements a day. He had been given bismuth preparation which apparently partially controlled the diarrhea. Several days later he developed an acute respiratory infection and the diarrhea returned.

Past History: He had had the usual childhood diseases but had never been sick for any extended period of time. He was always thin and never complained. He had always had an excellent appetite.

Examination revealed a white boy whose skin was sallow. There was pitting edema of both legs extending to the knees. The scrotum was markedly edematous and almost the size of a grapefruit. The prepuce was swollen and he had difficulty in voiding. The skin of the abdomen was tense and showed pitting edema. The hands and face were likewise swollen. There were no petechiae. The temperature was 99, pulse 120, respirations 18. The tonsils were large. There was a post-nasal drip with pus from the right ethmoid. The heart was slightly enlarged to the left to percussion. The sounds were regular and of good volume. There was a loud blowing systolic murmur present. The abdomen was tense and a fluid wave was present. The liver and spleen could not be felt. His weight was 83½ pounds.

Laboratory findings showed the total 24 hour urine output 1100 c.c., clear, specific gravity 1040, acid, no albumin, 6 per cent sugar, 2 plus acetone, no diacetic acid. The urine was negative for indican and showed 1 plus urobilin. Hemoglobin was 55 per cent, red blood cells 2,200,000, white blood cells 4,000. The smear showed basophiles 1 per cent, eosinophiles 1 per cent, myelocytes 1 per cent, juveniles 3 per cent, stabs 9 per cent, segmented 57 per cent, lymphocytes 25 per cent. The sedimentation rate was 35 in half an hour. The blood sugar was 171 mgm. per 100 c.c. of blood; serum protein 6.05. B.M.R. plus 4. The Wassermann was negative. The stool was negative for parasites or ameba.

It was obvious that this was a case of juvenile diabetes complicated by cirrhosis of the liver or chronic adhesive pericarditis associated with the rheumatic heart disease. During the following ten days an effort was made at establishing control of his diabetic condition and while this appeared to be under control he continued to gain in weight. Four days later his weight increased to 94½ pounds. He was given tr. digitalis until therapeutic effect was obtained. Weight loss was slow and on December 10 he was given 500 c.c. of whole blood. There was still no appreciable reduction in his weight. On December 16 he was given 1 c.c. salyrgan-theophyllin intravenously and again on December 20. The urinary output increased to 3000 c.c. From then on he began to lose weight. On January 5, 1944 he weighed 82 pounds. He was receiving 1900 calories with 15 units of protamine zinc insulin plus 15 units regular insulin for breakfast, 20 units regular for dinner and none for supper. With this regimen he continued to maintain his weight constantly and remained relatively free of sugar.

On February 26 his abdomen was found slightly distended and upon examination a large hard spleen was found which was freely movable and extended medially to the umbilicus and inferiorly to the crest of the ileum. In spite of the difficulty of controlling his diabetes and recurring episodes of edema, it was felt that a splenectomy was indicated. He received two transfusions preoperatively and was digitalized.

On March 13, 1944, under gas and ether anesthetic, a left mid-rectus incision was made expos-

ing a large, freely movable spleen which was easily delivered into the wound. All vessels were ligated in the pedicle and the spleen was removed without any difficulty. The liver was slightly enlarged and rusty brown in color. It was hard and showed a diffuse granular appearance characteristic of cirrhosis. He had a normal postoperative convalescence which was afebrile. The wound healed per primum and he was discharged on the seventh day.

Pathologic specimen showed a large spleen measuring 6" x 4" x 3". The edges were round and hard. The splenic notch was present. The spleen weighed 800 grams. The tissue cut with difficulty and the section showed the characteristic fibrosis present in Banti's splenomegaly.

On April 1, 1944 his weight was 80 pounds. His urine was free of sugar. His diet was maintained at 1900 calories and he returned to his preoperative insulin requirements. The liver edge was palpable three finger breadths below the costal margin.

Since his splenectomy he has never shown any edema and has been able to control his diabetes. He remained well until the beginning of 1945, when his diarrhea recurred. In April 1945 he began to lose weight. Stools which were almost watery in consistency were negative for ova and pathogens. Analysis of the stool indicated that the diarrhea was due to a fatty intolerance caused by atrophy of the pancreas. Large doses of pancreatic gland were necessary to control the diarrhea.

DISCUSSION

Juvenile diabetes is not as unusual as formerly believed. These cases, as in adults, are most frequently detected in the course of routine urinalysis. They are most difficult to control because allowances must be made for growth as well as the child's normal desire for sweets.

Splenomegaly is likewise not uncommon in childhood. The splenomegaly of Banti's disease, if there is such a syndrome recognized now, is probably the most frequent type of enlarged spleen. Cirrhosis of the liver at age 13 with ascites and generalized anasarca is, however, very unusual. A diligent search of the literature fails to reveal a similar case.

SUMMARY

A case report of a white male, aged 13, who has had known diabetes for two years and who coincidentally had a splenomegaly with cirrhosis of the liver, is presented. Splenectomy was performed because of the generalized anasarca and ascites without subsequent recurrence.

THE WATERHOUSE-FRIDERICHSEN SYNDROME

WITH A REVIEW OF THE LITERATURE AND AN ADDITION OF THREE NEW CASES FROM THE CHARITY HOSPITAL OF LOUISIANA

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HISTORY

Although Voelcker reported the first presumptive case of this disease in 1894, Andrews in 1906 was the first worker to describe a proved case of fulminating meningococcal septicemia, now commonly known as the Waterhouse-Friderichsen syndrome, in the literature.

In 1911, Waterhouse added one case and reviewed the 15 others in the literature up to that time. Seven years later Friderichsen described two of his own cases and collected 28 cases altogether.

Glanzmann, in 1933, was the first to apply the term Waterhouse-Friderichsen syndrome to this clinical picture. Three years later, Aegerter and Sacks reviewed the literature separately and collected 55 and 64 cases, respectively.

During the following years, among others, Kundstäedter added one case, Moss and Schenken two cases and Lindsay et al. seven cases, so that by 1943 Bernard and Jordan were able to collect 117 cases in the entire literature.

I have attempted to review the literature since that time and have collected 57 more cases: Rucks and Hobson, Potter and Bronstein, Hayes and Whales, Weinberg and McGarvack, Thomas and Leiphart, and D'Agati and Marangoni have each reported one additional case.

Day and Ikeda and Rosenthal have contributed two more cases each, while Strick has described three cases. Marangoni and D'Agati and Bush and Baley have added six new cases whereas Pratt et al. and Cosgriff have both reported four more cases. Finally Martland reviewed 19 new cases of Waterhouse-Friderichsen syndrome.

I have also attempted to review all cases of this syndrome at the Charity Hospital of

Louisiana during the past five years. During this period, only two cases were definitely proved at autopsy and one strongly suspected but not proved case of the Waterhouse-Friderichsen syndrome has occurred at this institution. Since these three cases have apparently not yet been added to the literature, they bring the total of reported cases up to 177, as far as could be determined.

ETIOLOGY

It is commonly agreed that the meningococcus is by far the most frequent causative organism of this disease-syndrome. Various authors believe that it is the offender in from 50 to 70 per cent of all cases. It must, however, be borne in mind that it is not the sole etiologic factor in all cases. The *Streptococcus hemolyticus* is probably the second most commonly isolated organism. Lindsay et al. demonstrated the presence of *Hemophilus influenzae* in both the heart's blood and the cisternal fluid in two of their cases. Other writers report *Staphylococcus aureus, pneumococcus, Bacillus coli, Bacillus pyocyaneus, Bacillus Friedländer* and *Neisseria flavus* respectively as the causative organism in their various cases.

PATHOLOGY

The most constant pathologic condition at autopsy is massive adrenal hemorrhage, occurring in 95 per cent of the reported cases, according to Aegerter. Usually as soon as the renal regions are exposed at autopsy, one is startled by the remarkable appearance of both adrenal glands which are commonly deep purplish red. They retain their normal shape but are increased in size and have tense capsules.

On section, each adrenal gland is the seat of massive hemorrhage which has usually destroyed almost the entire gland. On the other hand, in some cases only minute petechial hemorrhages can be demonstrated. When the bleeding is mild in degree, it is generally stated that the medulla is more involved than the cortex.

Under the hand lense it can be seen that in the middle zone (zona fasciculata) the hemorrhagic areas have a definite pattern of a great many parallel red streaks. In the inner zone (zona reticularis) the hemorrhages often have a lace-like or reticulated appearance.

In this connection it is of interest to note that according to Marangoni and D'Agati the patient's life expectancy is about inversely proportional to the extent of the adrenal damage; that is, the greater the adrenal hemorrhage, the shorter the clinical course and the less likelihood for the development of subsequent hepatic destruction.

Microscopic study reveals an enormous capillary distention of the middle and inner zones of the cortex. There is usually no evidence of inflammation, thrombosis or embolism. In most cases, the outer zone (zona glomerulosa) escapes except where it is involved in isolated, irregular areas. The capsule as well as the surrounding structures only rarely exhibit any evidence of involvement. If proper staining of the tissue is employed, in most cases many clumps of meningococci, or any other etiologic agents, may be seen throughout the cortex.

Nearly every case at autopsy presents an extreme cyanosis and lividity over the face, chest and lower extremities. Ecchymotic and petechial skin lesions are practically constant findings. These often also are seen in the conjunctivae. In about half the cases, small hemorrhages are present in the serosa of the peritoneum, pleura or pericardium. A few reports mention hemorrhage into the mucosa of the gastrointestinal tract. This, according to McLean and Caffey, may account for the abdominal pain and diarrhea sometimes seen. The brain usually shows congestion of the superficial vessels of the leptomeninges. Frank signs of meningitis are characteristically absent. This supports the observation made by Herrick, during the first World War, that most meningococcic infections are bacteremias before there is metastatic localization in the leptomeninges.

Too frequently to be incidental, hyperplasia of the thymus and the other lymphatic structures of the body is associated with this syndrome. For example, in report-

ing 11 cases in 1901, Little noted that Peyer's patches were much swollen and unusually prominent, "as in the early stage of typhoid", and that in addition the mesenteric glands were enlarged in six and the thymus in two cases.

The other viscera usually show no significant pathologic lesions, with the exception of acute cloudy swelling of the myocardium, the kidneys and the liver and terminal pulmonary edema.

PATHOGENESIS

Various theories have been proposed to explain the selective action of this disease on the adrenal glands. A few of these are as follows:

1. *Embryologic Interpretation*: The skin, adrenal medulla and nervous system all arise from ectoderm and are at the same time the chief organs affected by this disease. It has, therefore, been suggested by some workers that this is not an incidental finding but that the disease possibly may have an affinity for this embryologic layer.

2. *High Incidence of Status Lymphaticus*: The occurrence of the Waterhouse-Friderichsen syndrome, together with findings of status lymphaticus (if such an entity actually exists), has made some authors feel that there is some relation between these two.

3. *Venous Thrombosis*: Another theory states that thrombosis of the central veins in the adrenals leads to capillary distention due to backward pressure and thus causes the hemorrhages into the adrenal parenchyma.

4. *Arterial Thrombosis*: Symmetrical, bilateral thrombosis of the arteries of the adrenals is rather difficult to conceive, and therefore this theory is not widely accepted.

5. *Bacterial Embolism*: Although this is also a possibility, it is, however, not too likely that a great enough number of microorganisms should clump together in the adrenal blood vessels to cause actual obstruction to the blood flow.

6. *Effect of Meningococcic Toxins*: According to this theory, the toxin released by the causative organisms of the disease picture has a direct effect on the capillary endothelium and causes a rupture of its walls with subsequent hemorrhagic extravasations.

The question arises just what role the adrenal gland plays in the production of the Waterhouse-Friderichsen syndrome. Adrenal cortical insufficiency is suggested by:

1. The clinical picture of hypotension, dehydration and vomiting.

2. The laboratory findings of decreased blood sodium, and increased blood NPN, and occasional hypoglycemia.

3. The pathologic findings of adrenal hemorrhage.

Rogoff states that the experimental animal may suffer a loss of both adrenal medullas and one cortex without radically influencing his life and health. However, a bilateral removal of the adrenal cortex invariably leads to the animal's death. In some cases of the clinical syndrome under discussion the extent of the adrenal cortical destruction may be very small. On the other hand, it is well known that only a small portion of the adrenal cortex is necessary for carrying on the essential processes of life. Furthermore, there is only a small number of patients in Addisonian crisis who do not respond favorably to the administration of fluids, electrolytes and cortical replacement therapy. However, patients exhibiting the Waterhouse-Friderichsen syndrome often do not improve on a similar type of treatment. These factors have made some workers believe that hypoadrenalism is not the primary cause of death here, but that the exodus is actually caused by an overwhelming toxicity throughout the body.

Marangoni and D'Agati are of the opinion that the Waterhouse-Friderichsen syndrome consists of two distinct clinical stages: The first stage consists of circulatory collapse and usually leads to death. If the patient survives this stage, the second or hepatorenal stage begins; this is characterized clinically by marked oliguria with azotemia and pathologically by a severe toxic hepatitis associated with glomerular and degenerative tubular changes.

The hepatorenal syndrome was actually first described by Heyd in 1923 to explain

certain deaths following biliary operations Wilensky and Colp have advanced the most satisfactory theory thus far to explain this syndrome: they postulate the existence of an initial lesion which affects the hepatic cells primarily and the renal nephron secondarily.

Marangoni and D'Agati believe that adrenal hemorrhage is a predisposing factor in the development of hepatorenal failure and they advance the following hypothesis for this process: Destruction of the adrenal glands by hemorrhage throws the full load of detoxification primarily on the liver and secondarily on the kidneys, both of whose functions have already been depressed from the onset by shock and toxemia. The partially disabled liver is so overwhelmed by toxins that it is unable to perform its physiologic detoxifying function. As a result, the liver undergoes toxic necrosis and a potent tissue toxin is liberated which, together with the unneutralized bacterial toxins, flood the circulatory system. Finally, the kidneys attempt to rid the body of these harmful products of bacteria and deranged liver metabolism by excreting them through the proximal convoluted tubules which, during this process undergo degenerative changes. In addition, glomerular changes of the nephritic type occur.

An interesting sidelight is the fact that the toxins produced by the necrotic liver cells are actually harmful to the uninvolved hepatic tissue. This was proved by Boyce who injected water-soluble extracts of liver tissue from patients dying of the hepatorenal syndrome into dogs by the intraperitoneal route. The dogs all died when strong extracts were used, and hepatic and renal damage could invariably be demonstrated microscopically at autopsy.

MEDICO-LEGAL ASPECTS

Even though it is little recognized as such by the medical profession, the Waterhouse-Friderichsen syndrome often presents a real medico-legal problem. The reason for this is that death in this syndrome is usually extremely rapid (usually within 24 hours after onset of symptoms) and that it is therefore often considered to be of unexplained cause. Martland states that a good number of these deaths have to be investigated because of the suspicion of food or chemical poisoning, foul play or negligence. To illustrate this, a few examples of the résumés of some of his cases follow:

Case No. 1: Adult died in hospital 24 hours after vomiting mushrooms. Mushroom poisoning suspected.

Case No. 2: Adult died two minutes after admission to the hospital and 48 hours after onset of gastrointestinal symptoms. Relatives wanted to sue a "fish-market."

Case No. 3: Adult found dead in bed. Police suspected "mugging" or manual strangulation. Subject dead for two days and body in bad state of decomposition. Subject well when last seen alive.

SYMPTOMATOLOGY

The clinical manifestations of the Waterhouse-Friderichsen syndrome have not been generally recognized. This is probably due not to failure of diagnosis alone but also to the fulminating course of the infection and the scarcity of cases seen by any one observer.

By far the majority of cases occur in patients under one year of age. Lindsay et al. believe that about 90 per cent of all cases are seen in children from six months to nine years. Aegerter states that 70 per cent of the patients have been under two years. During the past few years, there have been many additional reports of this syndrome in adults. This, however, probably does not indicate an absolute increase of this syndrome in adults, but is believed to be due to a better system of case reporting in the armed forces during this war.

The patient typically states that he had enjoyed perfect health until the onset of the disease. It has been found by various workers that the only cases without sudden onset are those complicated by other conditions, which might have altered the characteristic early signs.

The initial symptoms may be quite variable. There may be restlessness, headache and malaise or there may be gastrointestinal symptoms, consisting of abdominal pain, loss of appetite, vomiting and pos-

sibly diarrhea. Fever of moderate or extreme degree may be present.

One of the outstanding clinical features is the development of cyanosis. This can usually be noticed first about 12 hours after the onset of the disease, and has frequently been reported even as the first clinical symptom. The cyanosis, being of such a severe nature, is often quite frightening to the relatives and physician alike. It colors the lips and fingernails of the patient dark blue and gives the skin a livid appearance. A few reports mention a peculiar alternation of cyanosis and pallor of the skin resulting in a peculiar mottled appearance.

Soon after the onset of cyanosis petechiae may be noted over almost the entire body. These spread with such extreme rapidity that their development can frequently actually be observed within several minutes. Lindsay, for instance, tells about a patient's mother who told that "the rash was not present when the child left the house for the hospital." The small petechiae present initially usually soon develop into large, violaceous purpuric areas giving the skin a blotchy appearance. Some of these areas are the size of a palm or even larger. They do not disappear on digital pressure. Sometimes these areas undergo necrosis and sloughing later.

When examining the patient at this time, one usually finds him to be desperately ill. The blood pressure is often too low to be determined and the pulse is weak or even imperceptible. The patient is in a state of shock and often is either stuporous or semicomatose. The examiner is impressed with the rapid progression of the disease from hour to hour, which Herrick has so well described by stating that "no other infection so quickly slays."

The extremities generally become cold and the body hot. The patient at this time also becomes incontinent. The respiratory rate greatly increases and respiration often becomes labored. Some patients develop stertorous breathing and typical Cheyne-Stokes respiration. Throughout this period numerous rales can be heard over the chest —at first of the fine crepitant and then later of the coarse, bubbling type. After frank pulmonary edema occurs, the patient usually goes into coma and dies shortly thereafter.

The occurrence of clinical evidence of meningeal involvement is variable. Some authors state flatly that it never occurs in a true Waterhouse-Friderichsen syndrome, while others believe that it is quite common. Many patients complain of violent headache at some time during the disease. The observation of abnormal reflexes, convulsions and various degrees of stiff neck have been recorded.

D'Agati and Marangoni believe that too little attention has been paid in the past to the initial anuria which was a constant finding in their cases. The anuria persisted for 24 to 36 hours and was replaced by a severe oliguria. At no time was adequate renal function reestablished, despite recovery from shock, evidenced by normal blood-pressure readings, the improved character of the pulse and the improved clinical appearance of the patient.

The temperature curve in most cases is of a septic, spiking type, which may range from subnormal to 108 degrees Fahrenheit. Early in the disease the rise in temperature is accompanied by a similar rise in the pulse rate, but as the disease progresses the cardiac rate increases more and more. The cardiac rhythm, however, is only rarely affected, but its quality is generally found to be poor and fluctuating.

There are a good deal of other more rarely encountered signs and symptoms found in the literature, some of which are: muscle flaccidity, low blood sugar, chills, tremor, cough, ankle edema, strabismus and rigidity of the abdominal muscles.

The entire course of the disease is usually unbelievably rapid and, as a rule, the patient expires within 24 to 48 hours after the onset. It can, therefore, easily be seen that it is essential to make the diagnosis as promptly as possible and to institute treatment at once, for if the development of the full clinical picture is awaited, the hope of survival is negligible.

LABORATORY DATA

The occurrence of positive blood cultures has been discussed above. In some cases the organism has been identified in the routine differential blood count. The probability of identification by this method is materially increased if the blood for the count is taken at the site of a purpuric cutaneous lesion. In some cases, leukocytes laden with suspicious diplococci may be observed by mere chance in the Wright's stained blood films. Humphrey and Toomey advocate the utilization of the "buffy layer" smears for gram-staining which permits a concentration of the invaded cells and organisms not possible in other methods. The limitation of the blood smear methods, of course, must be realized in that if the bacteremia is not heavy, it is not as satisfactory as would be a culture, and that in most instances it also lacks the positive identification made possible by culture. However, its use is of great value because by the time the cultural diagnosis is made, the use of chemotherapeusis may have been delayed too long already to be effective.

A mild to moderate leukocytosis with a relative increase of the polymorphonuclear leukocytes is present in most cases. A shift to the left in the cells of the myelogenous series was noted by Bamatter. Occasionally the occurrence of an extreme leukocytosis, with cell counts up to 100,000/cm. mm. has been reported. There is often a moderate reduction in blood platelets. The Leed-Rumpel test is usually positive. Determinations of the coagulation and bleeding time, as a rule, are inconsistent depending on the stage of the disease.

Reports concerning the blood chemistry in the Waterhouse-Friderichsen syndrome are still quite incomplete. It appears, however, that an increase in the blood non-protein-nitrogen, and a decrease of the blood sugar, blood sodium and blood cholesterol levels are fairly constant findings.

It is noteworthy that difficulty is often encountered in these cases in expressing enough blood for counts and smears by the usual needle-prick method and that venepuncture is also often impossible.

Urinalysis frequently reveals varying degrees of albuminuria and a fixation of the specific gravity. Hematuria and cylindruria have also been reported.

Examination of the cerebrospinal fluid reveals no constant alterations, either in the cytologic or chemical components. The causative agents of the disease may or may not be seen on smears. The cell count may be high enough to impart turbulence to the fluid but rarely, if ever, is the fluid frankly purulent. An increase in pressure is noted in a considerable number of patients.

DIAGNOSIS

The clinical picture of the Waterhouse-Friderichsen syndrome is usually characteristic enough for the correct diagnosis to be made. The most important thing is to keep the condition in mind in any critically ill child and to consider it in the differential diagnosis.

D'Agati and Marangoni list a number of points which they consider to be of diagnostic importance: petechial eruption rapidly becoming purpuric; severe shock; facial edema; anuria of 24 to 36 hours' duration; urinalysis showing fixation of the specific gravity, albuminuria, hematuria, or cylindruria; marked leukocytosis; isolation of the meningococcus by culture and, in some cases, by peripheral blood smear; renal failure, as evidenced by elevation of the blood non-protein-nitrogen and creatinine.

TREATMENT

The rational treatment of the Waterhouse-Friderichsen syndrome may be divided into the treatment of shock, toxemia, bacteremia and the administration of adrenocortical hormone substitution therapy.

The occurrence of shock in this disease may be due to a sudden incapacitation of the adrenal glands by hemorrhage. Suggestion for replacement therapy rests on this hypothesis. It may, however, also be due to the overwhelming infection. It is essential to keep these two possibilities in mind in combatting the state of shock.

The patient should be kept at a comfortable temperature. The free use of morphine sulfate and the placing of the patient in

the Trendelenburg position is also advocated. Parenteral fluids in quantities of 3000 to 4000 c.c. should be given cautiously over a 24 hour period. The intravenous administration of plasma is recommended by some because of its osmotic effect leading to the elevation of the blood pressure. Normal salt solution should not be given in excess, especially if pulmonary edema is present. The use of adrenalin in the treatment of shock is generally contraindicated, but an occasional 0.25-0.50 c.c. dose has been thought helpful by some clinicians.

Every possible effort should be made to combat the toxemia at as early a date as possible. Lindsay et al. believe that the meningococcus and *Hemophilus influenzae* are by far the two most common etiologic agents. If the invader cannot be immediately identified in a given case they, therefore, assume the presence of one of these organisms. In that case they suggest the routine administration of meningococcal antitoxin intravenously and anti-influenzal serum intramuscularly. The usual dose for the antimeningococcal serum is from 60,000 to 120,000 units within the first 24 hours. All precautions necessary for any form of intravenous serum therapy must, of course, be observed. Antitoxin should not be withheld while possible improvement from chemotherapy is being awaited since prompt neutralization of the toxin may prolong life and give chemotherapy the chance to rid the body of the organisms.

Chemotherapy has its greatest value in combatting the bacteremia. Sulfadiazine, which is the sulfonamide of choice, should be given intravenously at the beginning and its administration should be kept up by either this or the oral route in dosages great enough to maintain a blood level of 15-20 mgm. per 100 c.c. It is advisable to keep the urine alkaline at this time, either by giving 1/6 molar sodium lactate intravenously or by any other means, in order to decrease the chance for drug crystalluria.

It is advisable to augment the sulfonamide chemotherapy by the use of penicillin. Only little information is available on this subject in the literature until now. Weinberg and McGavack suggest the intrathecal administration of the drug initially in doses of 15,000 to 20,000 units, which is to be followed every three hours by the intramuscular injection of 20,000 to 40,000 units. In the very severe cases they believe that the drug should be given intravenously, preferably by the continuous-drip method.

Adrenal cortical hormone is given in the hope of combatting the adrenal failure caused by the hemorrhage. However, its value is still quite questionable. Nevertheless it should be given in adequate doses, as early as possible, and by the intravenous route. Bush and Baley recommend the use of a continuous infusion of 5 per cent glucose in saline, repeated intravenous injections of adrenal cortical extract and an occasional intramuscular injection of desoxycorticosterone.

The use of repeated small transfusions is frequently very helpful in combatting the hemorrhagic tendency. Vitamin K may be used empirically when a lowered percentage of prothrombin can be demonstrated.

Finally, the patient should have the benefit of all possible supportive therapy, such as temperature measures if the fever becomes excessive, sedatives if they seem indicated and oxygen when either cyanosis or respiratory distress are observed.

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CLINICO-PATHOLOGIC CONFERENCE

RALPH V. PLATOU, M. D.*
RALPH G. SMITH, M. D.†
AND
CHARLES E. DUNLAP, M. D.‡
NEW ORLEANS

The following is a clinico-pathologic conference presented to the combined Junior and Senior classes at Tulane Medical School.

From the Departments of *Pediatrics, †Pharmacology and ‡Pathology of the School of Medicine, Tulane University, New Orleans.

PROTOCOL

P. J., a colored female, aged 15 months, was admitted to the hospital at 2:15 p. m. on November 15, 1945 and died at 9:45 p. m. on November 15, 1945.

C. C.: **The patient drank one-half glass of kerosene 45 minutes before admission.**

P. I.: **Mistaking it for water, the mother gave the child a glass of kerosene to drink. She believed the child took four to six ounces, then became unconscious, and within five minutes there was foaming at the mouth, gasping, irregular respirations, and frequent coughing. On the advice of a neighborhood druggist, the mother poured a quarter glass of milk and a teaspoon of castor oil into the patient's mouth, causing more coughing. Coma persisted, but there were no convulsions. Up to this time the child had always been well.**

P. E.: **T. 101.4°. A very well developed and well nourished colored female infant in deep coma, from which she could not be aroused. There was a distinct odor of kerosene on the patient's breath. No cyanosis or edema were noted. Respirations were rapid but of good quality. Coarse rales were heard throughout both lung fields. Corneal and gag reflexes were intact, and no pathologic reflexes were elicited.**

Laboratory: **Hemoglobin 10.0 Gm., white blood cells 10,800 with 88 per cent lymphocytes, 8 per cent polymorphonuclear cells, 2 per cent eosinophiles, and 2 per cent monocytes.**

Course: **Immediately after admission she was given 25 c.c. of 50 per cent glucose intravenously. A generalized convulsion lasting two and one-half minutes occurred without cyanosis. Oxygen was started by a nasal tube, and an infusion of 10 per cent glucose in water was continued for the next six hours at the rate of 50 c.c. per hour.**

Four hours after admission a similar dose of 50 per cent glucose was given, followed by subcutaneous injection of sodium sulfadiazine. Five hours after admission a milder generalized convulsion lasted for two or three minutes with no change in the respiratory pattern.

Dyspnea was first obvious at six and one-half hours, with occasional deep inspirations separated by periods of apparent hypopnea. Abdominal distention was poorly controlled by prostigmine. Repeated aspirations of upper respiratory tract yielded only small amounts of mucoid material from the posterior nasopharynx; metrazol and artificial respiration during the last hour of life did not affect the increasing respiratory difficulty.

Dr. Platou: I am sure most of you would believe a case of kerosene poisoning such as this one seems too obvious to require much elucidation in a clinico-pathologic conference. Unfortunately, there are lots of loopholes in our knowledge of effects of such

poisoning, justifying a good deal of elucidation and suggestions for improving our knowledge.

Kerosene, as you all remember, is a mixture of more or less volatile hydrocarbons, and, as you know, the hydrocarbons have certain properties in common. They act in some situations as narcotic agents, and many of them are of inestimable value in anesthesia. Others act as volatile asphyxiants, and most are chemically inert in the human body. Kerosene, being a mixture coming off at a given stage in the process of distillation, undoubtedly varies in composition from lot to lot and distillery to distillery. There is no valid method used for estimating the degree of absorption or to correlate intoxication, asphyxia, or narcosis with variable amounts of such absorption. There are a few things that we do know: Enough cases have been studied in humans, dogs, and rabbits to prove conclusively that there is no constant effect on the blood morphology or constituents which have been measured in clinical laboratories. In a few cases there is a transient acetonuria; a few cases of methemoglobinemia have been reported, but in larger series this finding has been notably inconspicuous. If one gave experimental animals stated doses of kerosene of known composition by various routes and followed the iodine numbers of the serum fatty acids, he might indirectly relate degree of absorption to observed effects. This has not been done, however, as far as I have been able to determine. Because the narcotic properties of a hydrocarbon increase with its volatility or in inverse proportion to molecular weight or its solubility and because kerosene is made up of a mixture of varying molecular weights, volatility, and so on, it is not surprising to note that its effects in humans as well as in experimental animals have been variable.

When we started our work here, we laid down a rule for our service as far as kerosene is concerned—that no stomach tube should be used. I have no reason to change the rule because of this case. So far as I can gather from experimental work and reported human cases, deaths rarely if ever occur unless there has been aspiration. In animals the lethal dose of kerosene has been upwards of 20 c.c. per kilogram given by stomach tube. Given intratracheally, amounts as little as 0.5 to 0.75 c.c. may be lethal within a very few hours. If the patient has just swallowed kerosene and has none in the tracheobronchial tree, there will usually be only a relatively profound narcosis with recovery. If the kerosene were both ingested and aspirated, the harm has already been done and there is no means of effectively removing the hydrocarbon from the tracheobronchial tree by mechanical means. If the child has not already aspirated, then the passage of a stomach tube with the usual incidental struggling, gagging, and vomiting is certainly a good way to induce aspiration.

All the collected observations in experimental animals and humans certainly justify the contention that the single most important prognostic feature is the amount of hydrocarbon in the lungs; mortality rate is related directly to the amount of aspiration.

There are other effects with ingestion of kerosene, a rather violent gastroenteritis, with nausea, vomiting, and diarrhea being common. These may be masked to some degree by the narcosis. In the tracheobronchial tree there is violent inflammatory edema and congestion of the alveolar capillaries with rupture of a few. I have here a report of a case in a 33 year old seaman who drowned in gasoline, which describes similar pulmonary manifestations. It seems surprising to note that among 133 cases in three series, in addition to a few individual case reports, there have been no good descriptions of pathologic findings in the brain, nor have spinal fluid examinations been recorded; yet many of these cases have symptoms suggesting meningeal irritation.

Many events observed clinically after kerosene ingestion are illustrated quite typically here. Aspiration, as in this child, is evidenced by coughing and gagging immediately after ingestion. There are convulsions in 2 to 3 per cent of the cases. The critical period during which most deaths occur is also well illustrated here—within

twelve to fourteen hours. Aspiration in this case was perhaps further encouraged by the druggist's advice to administer milk and castor oil to a child already comatose and unable to swallow; we may see castor oil and milk curds in the lungs in addition to the kerosene pneumonitis. The child weighed 22 pounds, and if the mother's estimate of 140 to 150 c.c. kerosene ingestion is correct, she may have received a lethal dose by ingestion alone but had plenty of opportunity for far more than a lethal dose by aspiration.

Regarding treatment, we are faced with the situation in which the narcosis is perhaps due to a combination of asphyxial and histotoxic anoxia; there are only a few things that can be done. First, let me say gastric irrigation should be avoided, as it enhances the chances of further aspiration. Second, I think with the most obvious feature being deep narcosis, these patients should be stimulated by chemical or mechanical means. Because they are asphyxiated to some degree, oxygen or oxygen-carbon dioxide mixture should be given. For irritative changes in the gastrointestinal tract, demulcents and absorbents should be used. Kerosene itself acts as a violent cathartic agent. While the patient is in coma, of course, extreme care must be used to avoid aspiration, and it is perhaps best to give nothing by mouth until he is revived.

The next point should be prophylaxis against the less frequent deaths beyond the acute stage, employing sulfonamides against the usual pyogenic invaders. If death does not occur in twelve hours, the patient is almost always out of danger except for the possibility of a complicating pneumonia, bronchiectasis, or lung abscess. Sulfonamides should be begun prophylactically as soon as the patient has recovered from the narcotic state, or perhaps earlier if given by parenteral routes. This patient, for example, was given sulfadiazine subcutaneously within four hours after admission. Hypertonic fluids may be useful in combatting the violent inflammatory pulmonary edema. This child had evidences of pulmonary edema and may have had cerebral edema as well. The amount of fluid given this patient may be open to criticism. It was not excessive—50 c.c. per hour for six hours. She also received two 25 c.c. doses of 50 per cent glucose. I suppose in the face of obvious pulmonary edema the parenteral administration of fluid could be criticized; here it was used to promote diuresis and reduce pulmonary edema. I think it was justified.

Breathing was of the Cheyne-Stokes type, deep gasps with intervening hypopnea as commonly seen in metabolic alkalosis and in cases of terminal diseases of the central nervous system.

In summary, I believe that nothing was done in management of this case that deserves serious criticism. The administration of 350 c.c. of fluid was not excessive for this patient; faced with the same situation again, we might wish to modify treatment to include more vigorous use of stimulants until narcosis had passed. With this addition, however, I doubt if the outcome could have been improved.

Dr. Rollings: You mentioned that only a small amount of kerosene was necessary to produce death—less than 1 c.c.?

Dr. Platou: Yes, by aspiration. We have no data about the rates of absorption, but it seems obvious that they, as well as the inflammatory changes, occur most rapidly in the respiratory tract.

Student: What do you think about lack of cyanosis in view of the asphyxia?

Dr. Platou: The only answer I have for that is that the patient was colored, and it is often difficult to detect cyanosis in the colored race.

Student: Do you think aminophyllin would have any effect on such narcosis?

Dr. Platou: I doubt it.

Dr. Ray: How easy is it to get an infant of this age to drink kerosene?

Dr. Platou: Surprisingly easy, apparently.

Dr. Dunlap: Dr. Ralph Smith has been kind enough to come in to take part in the discussion of this case.

Dr. Smith: Everything I have to say has already been said, so I will take little time. In these cases gastrointestional manifestations, which are not usually serious, are often experienced. Patients who can describe their symptoms complain of burning pain in the stomach, which may or may not be severe. There may be nausea and vomiting, but it is my impression that vomiting does not always occur. Further irritation of the gastrointestinal tract may cause diarrhea. These effects are probably unimportant as a cause of death.

On absorption, there are actions on the central nervous system. Early, there may be intoxication, more commonly seen with lower boiling point hydrocarbons than with kerosene. This recembles alcoholic intoxication, including delirium and depression of the respiratory center. Occasionally pryexia occurs. There are a few cases recorded with temperatures of 105°F. or 106°F. This patient showed convulsions, which are not uncommon but in the majority of cases they do not appear.

The heart is sometimes affected, as indicated by a rapid, weak pulse. Whether this is caused by a direct action on the heart or is secondary to anoxia, I do not know. I have no explanation for the cherry color of the blood. Methemoglobin has been described but is not a constant finding.

As Dr. Platou has already emphasized, aspiration greatly increases the toxicity of kerosene. In dogs it is from ten to twenty times as toxic administered intratracheally as it is when administered by stomach tube. In one report of 65 cases of kerosene poisoning with six deaths, in all fatalities there was lung involvement, which was assumed to mean aspiration. Of the 59 recoveries, there was pulmonary involvement in 32 per cent. Whether it is possible to have pulmonary involvement without aspiration, I do not know. I think it is much more probable that this would take place with lower boiling point hydrocarbons, such as gasoline or benzene. Kerosene has a boiling point of 150° to 300°C., so that it is not very volatile and the amount excreted by the lungs would not be very great. Gasoline would be excreted by the lungs to a greater degree and would more probably produce lung changes without aspiration.

I am not sure of the cause of death in kerosene poisoning. Probably the best guess is depression of the respiratory center. Again this is undoubtedly contributed to by local irritation of the lungs, causing inhibitory effects on respiration. Aside from any specific effect on the center it is very easy in animals to produce death by putting liquid in the lungs. Fluids going into the trachea instead of the esophagus often cause immediate death. This cannot be entirely due to blocking of the lungs, because it does not take that much fluid. Reflexes probably play a role.

Gastric lavage involves some danger, as Dr. Platou has stated. However, it has not always been considered wise to omit it. In this series of 65 cases which I mentioned, the authors advised gastric lavage, and it was carried out in almost all cases in spite of the fact that they realized that kerosene is much more toxic when aspirated and also that aspiration may take place as a result of passing the stomach tube. They stated that administration of emetics was preferable because it avoided aspiration. Again I have nothing to add as far as lethal dose is concerned. In most cases the amount taken was not known. One-third of an ounce or less has been reported as fatal. Dr. Platou mentioned one case in which 750 c.c. was taken without death. McNally reported one in which a liter was taken with recovery. The variable tolerance may be explained by differences in the rate of absorption and corresponding differences in concentration in the blood stream and brain at any one time. The toxicity of kerosene absorbed from the gastrointestinal tract may be further modified by metabolic changes in the liver, but that is purely speculative.

Dr. Dunlap: This child at the time of autopsy was well developed and adequately if not excessively hydrated. There was edema of both feet and slight subcutaneous edema. When the body was turned over, fluid ran from the nostrils. There was not

an excessive amount of fluid in the pleural cavities.

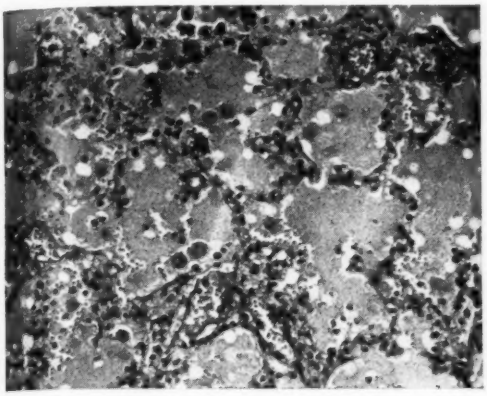

Fig. 1. Photomicrograph of a characteristic portion of the lungs. The alveolar spaces are completely filled with edema fluid except for a few minute bubbles of air. Occasional macrophages have wandered out into the edema fluid. The alveolar walls and other tissues of the lung show no evidence of serious injury.

The lungs were very wet and frothy, and fluid poured from them when they were cut. There was no crepitant lung tissue present except for scattered, small patches; the lungs were thoroughly water-logged. Very little of note was found elsewhere in the body. The stomach was distended with gas and contained about 50 c.c. of white material which looked like milk. There was no gross evidence of injury or hemorrhage in the gastrointestinal tract. The odor of kerosene was easily detected in the contents of the stomach and in the small and large intestines and also in the lungs. Although this is a crude test, I believe it is reliable. The liver showed no gross change except for its yellow color. Special stains revealed a moderate amount of fat in the liver cells. A child dying of almost any disease might have as much fat as was seen here. The heart was not abnormal except for thickening of the endocardium, particularly in the left atrium. Dr. Arey made a careful examination of the brain with routine stains and found no abnormalities. The spinal cord showed no changes. The kidneys showed some degeneration of the epithelium of the convoluted tubules and diffuse hyperemia. The quantity of fluid administered to this child is not clear to me; Dr. Platou may have some inside information, but it is difficult to tell from the chart how much the child received. An infusion of "1000 c.c." was started shortly after admission, and I cannot tell from the chart when it was discontinued. Another "1000 c.c." was started five hours before death and again we do not know over what period of time or at what rate it was given. We have no clear evidence that fluids were administered in excess but we do know that at the time of death the child had massive edema of the lungs and moderate edema of the feet. I believe it is fair to say that the child had adequate fluid and probably too much.

In my opinion this child died of pulmonary edema rather than from any cerebral effects of kerosene poisoning. Lungs that contain almost no air at all and are filled with fluid give us an adequate cause of death without seeking other causes. I do not know the amount of parenteral fluid given, but whatever it was I doubt that it helped the patient and it certainly could have contributed to the development of pulmonary edema.

I do not know of any effective specific treatment for kerosene poisoning but from what has been said by Dr. Platou and Dr. Smith it is clear that control of pulmonary edema might save a good many lives. An article by Hardy and Barach* in the June second issue of the Journal of the American Medical Association discusses the effectiveness of positive pressure respiration in the treatment of pulmonary edema. I would like to read a brief excerpt from their article in order to support a suggestion that positive pressure respiration might have been used to advantage in the case we are discussing today.

"Carlisle reported that the administration of oxygen with the mask which provided pressure in expiration was a most effective method of treating and preventing

*Hardy, G. C., and Barach, A. L.: Treatment of pulmonary edema due to chlorine gas, J. A. M. A., 128:359, 1945.

pulmonary edema due to irritant gases. Rovenstein described a case of irritant gas poisoning in which acute pulmonary edema was successfully treated by the Meter mask, which provided positive pressure in expiration. Segal has also reported the favorable effects of positive pressure respiration in acute pulmonary edema.

"The physiologic basis for employing positive pressure in the treatment of irritant gas poisoning is in the main the application of a direct physical pressure on the external surface of the pulmonary capillaries. Approximately 50 per cent of the applied intrapulmonary pressure is lodged on the capillary wall, the remainder being taken up by the elasticity of the expanded lung. Although positive pressure in both cycles of respiration is a more effective method, the application of positive pressure during expiration alone is simpler."

Positive pressure respiration is relatively safe and may do a great deal to relieve pulmonary edema. Since in kerosene poisoning, survival for twelve hours often means permanent recovery, control of acute pulmonary edema can be a life-saving measure. Lesser, Wiener, and McCoy report that the important pulmonary changes in kerosene poisoning are edema with superimposed pneumonitis. The autopsy findings in this case are in keeping with their observations.

NEW ORLEANS
Medical and Surgical Journal
Established 1884

Published by the Louisiana State Medical Society under the jurisdiction of the following named Journal Committee:

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SUBSCRIPTION TERMS: $3.00 per year in advance, postage paid, for the United States; $3.50 per year for all foreign countries belonging to the Postal Union.

News material for publication should be received not later than the eighteenth of the month preceding publication. Orders for reprints must be sent in duplicate when returning galley proof.

Manuscripts should be addressed to the Editor-

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CHANGES AT LOUISIANA STATE UNIVERSITY, SCHOOL OF MEDICINE

When Dr. Chester Stewart became Director of Pediatrics at L.S.U. in the summer of 1941, the pediatric group in the South was much enriched and improved by the accession of this modest, capable man, who speedily endeared himself to all with whom he came in contact. Dr. Stewart was strictly the product of the Middle West, having received his training in the great state universities of the northern Mississippi Valley. He was keenly interested in research, particularly in the field of tuberculosis in children. It is not generally known that Dr. Stewart had an exceptionally broad training aside from pediatrics, including both anatomy and pathology. In anatomy he was so well qualified that when an emergency made it necessary to name an Acting Head of the Department of Anatomy at L.S.U. there was no hesitancy in asking Dr. Stewart to assume this added responsibility. All this was accomplished although Dr. Stewart's health was by no means good, much as his appearance belied this. A long standing cardiac condition unfortunately terminated the fruitful and promising career of this valuable man on February 8, 1946.

When the authorities of L.S.U. began to cast about for a worthy successor to Dr. Stewart there was found among pediatricians and medical educators an almost unanimous opinion that the best possible pediatrician to fill the vacancy would be Dr. Myron E. Wegman, a young man connected with Cornell and Johns Hopkins in academic work and with the New York City Department of Public Health as director of training and research in child hygiene and director of school health.

In contrast to Dr. Stewart, Dr. Wegman is a product of the northeastern universities, College of the City of New York; Yale, where he took his degree in Medicine; and Johns Hopkins, where he took a graduate degree. While Dr. Wegman's interest is primarily in clinical pediatrics he may be expected to stress the preventive aspects of the subject—a field to which he has devoted so much time and interest. We may safely predict for Dr. Wegman a brilliantly successful career in his new field of endeavor.

The pediatric department is also fortunate in that the new dean at L.S.U. is a pediatrician. Dr. Vernon W. Lippard, recently associate dean at the College of Physicians and Surgeons of Columbia University, took both his undergraduate and

medical degrees at Yale and received his pediatric training in New Haven and New York. During the war he served in the South Pacific and held the rank of Colonel on return to civilian life. Although administration of the affairs of the School of Medicine will be his chief responsibility, Dr. Lippard will participate actively in the work of the Department of Pediatrics and will bring to it the benefit of his outstanding experience in research and teaching.

G. W. McC.

ACCIDENTAL POISONING OF INFANTS AND CHILDREN

Although no individual physician encounters very many examples of acute poisoning in his daily work, the magnitude of this problem in hospital practice is great. We have been impressed by the large number of infants and children dying in our hospitals each year as a result of such avoidable accidents as aspiration of kerosene, ingestion of roach paste, or corrosive esophageal strictures due to lye.

All would agree that prevention is the only logical solution to this important problem. Physicians must necessarily assume a leading role in any effective continuous educational program for this purpose. Specifically, any physician caring for children should count among his responsibilities the duty of warning parents of the common sources, nature, and effects of at least the most common poisons. Just as mass immunization programs for infants and preschool children have materially reduced hazards from many common communicable diseases in these age periods, so can conscientious propaganda reduce morbidity and mortality from such even more preventable causes.

Effective therapy in many true emergencies is often delayed by failure to identify promptly particular toxic agents or by injudicious hasty measures so freely advised by well-meaning neighbors and friends. Recommendations for immediate treatment of particular poisonings posted in the accident rooms of many large hospitals are often outdated and sadly in need of revision.

We should like to make this an editorial plea that all practicing physicians review their readily available references concerning most recent advances in therapy and form appropriate committees to prepare up-to-date recommendations for guidance of their own hospital staffs. Having a background of experience with the consequences of common poisonings, pediatricians should be best equipped to institute sensible campaigns for prevention and proper therapy.

M. L. W.

PEDIATRIC NUMBER

This is the second December issue of the Journal that has been devoted exclusively to pediatric subjects. The first pediatric number was made up of contributions from the Departments of Pediatrics of Tulane and Louisiana State University Medical Schools. At that time the late lamented Dr. Stewart, who was Professor of Pediatrics in his school, wrote an editorial in which he expressed hope that articles for later issues would be submitted by physicians who are particularly interested in diseases of children and of childhood. This second pediatric number has fulfilled the wish of this teacher of pediatrics. Merely taking the first article in this journal, it will be seen that it is an outstanding contribution by a group of pediatricians from Shreveport. It is a truly splendid article in every respect and a superb study of infectious hepatitis. There are other papers as well that have come from men who are not connected with the Departments of Pediatrics in the medical schools which will merit reading and attention.

It is believed that the publication of the specialty number has helped to incite interest in the particular specialty of pediatrics. It might be that other specialties should have special numbers and that, in this way, the general practitioner would

feel stimulated to report on some of his interesting cases or an unusual case and that the specialists themselves might also be glad and willing to contribute and to make possible a specialty number.

WHAT ARE THE FUNCTIONS OF A CANCER DETECTION CLINIC?

The development of two cancer detection clinics in New Orleans, one at Tulane and one at Louisiana State University, makes it necessary that we, as physicians, all know and understand the functions of such a clinic and our own relationship to it.

Cancer detection clinics are for *presumably well* individuals. Those patients who have symptoms are expected to consult their own physicians about their complaints. If the physician then suspects the presence of a malignancy, the patient is *not* to be sent to the detection clinic, for the function of the clinic has been fulfilled as soon as a physician suspects the presence of cancer. If the physician seeks aid in final diagnosis, the patient should be sent to another physician, who, in the opinion of the patient's physician, is capable of rendering help through special technics, or to a cancer diagnostic clinic, if one exists in that area and the referring physician elects to use it.

When the detection clinic suspects cancer, the patient is sent back to the referring physician or to a diagnostic clinic, so that biopsy can be done. Biopsies are not done in detection clinics, according to the regulations of the American Medical Association.

Because cancer detection clinics do not presume to diagnose already suspected malignancy, no emergency service has been set up for seeing patients who are suspected of having malignancies by signs or symptoms. Patients are examined in order, as they present themselves, and, as a result of the interest aroused in cancer, one of the clinics is at present about seven weeks behind in appointments. No admitting service is provided to note immediate needs of patients, for presumably there are none.

Physicians, therefore, must know and understand the functions of such detection clinics, so that patients requiring immediate care are not erroneously placed on such waiting lists. The admitting personnel of the detection clinics has done a good, but probably not a perfect, job of keeping this relationship straight. A thorough understanding by the physician is also necessary in order that he, as well as his patients, may obtain the maximum benefits from such clinics.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

1947 ANNUAL MEETING PROGRAM

At the call of the President and in accordance with instructions of the Executive Committee, a meeting for consideration of plans for the scientific program for the 1947 meeting was held in the office of the Society on November 21. Seventeen doctors, including the President, the Secretary, Chairman of the Committee on Arrangements, members of the Committee on Scientific Work and Committee to Study Rearrangement of Annual Meeting Programs and Chairmen of Scientific Sections were present.

After considerable discussion motion was made and carried that the general plan as outlined at the 1946 meeting of the House of Delegates be followed, that a clinico-pathologic conference be held on the first night and three or four symposia on

the second day, with half hour periods for questions, and the last day devoted to special papers; leaving to the general chairman in charge of the program arrangement and number of symposia. It was the opinion of the doctors present that if the executive officers see fit a meeting of the House of Delegates could be held on Sunday and continued on Monday morning and a symposium held in the afternoon on Monday.

It was brought out in the discussion that the special papers to be presented on the last day would include papers secured by the chairmen of the various sections, these papers to be presented either before special groups or the general membership, as the sectional chairmen may indicate.

The President, subsequent to the meeting, appointed Dr. Donovan Browne to serve as general chairman of the scientific program for 1947. It was brought out in the meeting that the general chairman should notify the chairman of each section concerning subjects selected for the symposia so that essayists may be selected accordingly. It was felt that special subjects could be rotated, to include one or two each year.

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SPECIAL REQUEST TO MEMBERS

Recently every physician licensed to practice in the State of Louisiana was mailed a brochure, explaining the new prepayment surgical and obstetrical care plan, known as Louisiana Physicians Service, Inc., sponsored and approved by the Louisiana State Medical Society. Accompanying the booklet was a Participating Physicians' Agreement with a letter requesting the doctor to review the booklet, sign the card agreeing to participate in the prepayment surgical plan and support the Society's efforts to defeat socialized medicine by cooperating and making this undertaking a success.

The enrollment of Participating Physicians is in charge of Dr. A. V. Friedrichs, Chairman of the Council on Medical Service and Public Relations.

A review of the participating physicians cards as of November 15, revealed approximately thirty (30%) per cent of the doctors signed the agreements and agreed to cooperate with the Society in the promotion of the prepayment plan. Some have written to the office explaining they were in fields other than surgery and obstetrics, and therefore not covered by the plan. It is the ultimate desire of the Society to include every field of medicine in the plan; therefore, we again ask each doctor to sign the participating physicians agreement even if at this time he can receive no benefit from the plan. It is the Society's desire to be able to report one hundred per cent participation by the doctors.

This is your plan. What you do with it, and about it, will determine the extent to which it will succeed. If you study it, and acquaint yourself and your patients with it, and use it properly, it will help you, help your patients, and make Federal compulsory medicine unnecessary. If you ignore it, abuse it, or fail to participate in the plan, you will be doing all you can to make Federal control of medicine a certainty.

Last month's issue of the New Orleans Medical and Surgical Journal contained a complete description of the plan including the schedule of fees payable for services performed. It is true some of the fees are low and others need adjustment. We acknowledge this with the statement that as soon as experience is gained and reserves established adjustments in the fee schedule will be forthcoming to increase the benefits.

If you have not received the material explaining the plan in the mail, please refer to the last issue of the Journal or write to Louisiana Physicians Service, Inc., Room 103, No. 1430 Tulane Avenue, New Orleans, for additional information.

For those who did not receive, or have misplaced this participating physicians' agreement, a copy of the agreement accompanies this article.

Please cooperate with the State Society by signing the following participating physicians' agreement and return it to the Society's office.

PARTICIPATING PHYSICIANS' AGREEMENT

I, the undersigned, a Doctor of Medicine licensed by the Louisiana State Board of Medical Examiners, in consideration of being accepted as a Participating Physician of Louisiana Physicians Service, Inc. herein termed the Corporation, do hereby agree with the Corporation as follows:

I will perform the surgical and/or obstetrical services specified in the Certificates issued by the Corporation for an individual subscriber whose annual income does not exceed $2,000.00 or to a family group subscriber whose annual income together with the income of his spouse and/or family members, does not exceed $3,000.00, at the rates of compensation set forth in the Schedule of Benefits, and will make no charge to the subscriber for such services. In the event services are rendered to an individual subscriber whose annual income exceeds $2,000.00 or to a family group subscriber whose annual income, together with the income of his spouse or family members exceeds $3,000.00, I will accept the indemnity set forth in the Schedule of Benefits as full payment of liability from the Corporation, for services rendered, and any difference between the indemnity paid and my usual fee may be charged to the subscriber.

Thirty (30) days' written notice of termination of this agreement be given by either party.

There shall be no change in the usual patient-physician relationship except in the manner of payment for services under the Certificate and I remain free to act in relations with my patients in accordance with the custom in ordinary practice.

Accepted and Agreed at
New Orleans, Louisiana:
Louisiana Physicians Service, Inc.

Signed:

.....M. D.

Date.....Address.....

By.....

.....

—o—

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

TRANSACTIONS OF THE ORLEANS PARISH MEDICAL SOCIETY

CALENDAR OF MEETINGS

Dec. 9 Scientific Meeting, Orleans Parish Medical Society, 8 p. m.

Dec. 11 Woman's Auxiliary, Orleans Club, 3 p. m.
Touro Infirmary Staff, 8 p. m.

Dec. 12 Clinico-pathologic Conference, Touro Infirmary, 12 noon.

Dec. 13 Executive Committee, Hotel Dieu, 8 p. m.

Dec. 16 Hotel Dieu Staff, 8 p. m.

Dec. 17 I. C. R. R. Hospital Staff, 12:30 p. m.
Baptist Hospital Staff, 8 p. m.
Charity Hospital Medical Staff, 8 p. m.

Dec. 18 Clinico-pathologic Conference, Marine Hospital, 7:30 p. m.
The Orleans Society of X-Ray Technicians, 7:30 p. m.
Charity Hospital Surgical Staff, 8 p. m.
French Hospital Staff, 8 p. m.

Dec. 26 Clinico-pathologic Conference, Touro Infirmary, 12 noon.
DePaul Sanitarium Staff, 8 p. m.
Lakeshore Hospital, 8 p .m.

Dec. 27 L. S. U. Faculty Club, 8 p. m.
New Orleans Hospital Dispensary for Women and Children Staff, 8 p. m.

Jan. 6 Board of Directors, Orleans Parish Medical Society, 8 p. m.

Jan. 7 Orleans Parish Radiological Society, 7:30 p. m.
Eye, Ear, Nose and Throat Staff, 8 p. m.

Jan. 8 Woman's Auxiliary, Orleans Club, 3 p. m.
Touro Infirmary Staff, 8 p. m.

Jan. 9 Clinico-pathologic Conference, Touro Infirmary, 12 noon.

Every Thursday, Medical Toastmasters, New Orleans Athletic Club, 6:30 p. m.

RADIO PROGRAM

The following "Health Discussions" were presented over radio station WDSU during the month of November:

November 4—The Eye, Dr. John Gooch.

November 11—Epilepsy, Dr. J. A. Colclough.

November 18—Stomach Ulcers, Dr. Jules Myron Davidson.

November 25—Deafness, Dr. Mercer G. Lynch.

Our radio programs for the year 1946 came to

a close with the November 25 presentation. Time given to us by station WDSU has been withdrawn because of other commitments.

NEWS ITEMS

The following members of the Society attended the Fourteenth Annual Meeting of the Central Association of Obstetricians and Gynecologists in Chicago, September 12-21:

Drs. W. D. Beacham, John S. Herring, C. Gordon Johnson, Walter Levy, Harry Meyer, and Thomas B. Sellers.

At this meeting Dr. Beacham presented a paper on "The Management of Large Uterine Myomata with a Report of a 55 Pound Tumor."

Dr. Hunter L. Harang attended the Twenty-first Annual Congress of Anesthetists in New York, September 9-13. Dr. Harang presented a paper on "A Combination of Sodium Pentothal and Nitrous Oxide for Oral Surgery."

Dr. B. Bernard Weinstein attended the meeting of the Alabama State Association of Obstetricians and Gynecologists in Birmingham, October 17. At this meeting Dr. Weinstein discussed a paper on "Sterility."

CHANGES IN MEMBERSHIP ROLLS

During the month of October the following changes took place in our membership rolls:

Dr. H. B. Alsobrok, an active member, was elected to honorary membership.

Two associate members (Drs. Mayo L. Emory and N. Leon Hart) were transferred to active membership.

One active member (Dr. Jos. M. Montagnet, Jr.) and one member recently returned from military service (Dr. Emile A. Bertucci, Jr.) were elected to inactive membership because of their temporary removal from New Orleans.

Dr. Gilbert Balkin, recently separated from military service, was removed from the rolls because of his removal to Denver.

CHANGES IN ADDRESS

Dr. Irvin Cahen, 3705 Prytania Street

Dr. J. A. Colclough, 406 Marine Building.

Dr. Wilfred Finkelstein, 1320 Foucher Street.

Dr. Samuel Karlin, 204 Medical Arts Bldg.

Dr. Louis K. Levy, 3705 Prytania Street.

Dr. Margaret A. Maynard, 1318 Foucher Street.

Dr. Joseph Stamm, 1427 Delachaise Street.

SEPARATED FROM MILITARY SERVICE

The following physicians were recently separated from military service:

Dr. Emile A. Bertucci, Jr., St. Elizabeth's Hospital, Bogalousa, La.

Dr. Joseph L. DiLeo, 1903 So. Dupre Street.

Dr. Jack R. Hays, Charity Hospital.

Dr. Gilbert Balkin, 1003 Republic Building, Denver, Col.

Dr. Warren Hebert, 810 Hibernia Building.

LIBRARY NEWS

Members of the Society who read Italian will be interested to know that the valuable files of the Italian journal Policlinico have been presented to the Matas Library by Dr. Rudolph Matas on the occasion of his 86th birthday.

This journal is published in three sections, (Sezione Chirurgica, Sezione Medica, and Sezione Pratica), and contains publications representative of the best medical thoughts of Italy for the past 50 years.

NOMINATED FOR OFFICE

At the regular meeting of the Society held Monday, November 11, 1946 the following nominations for Officers of the Orleans Parish Medical Society for 1947, and Delegates to the Louisiana State Medical Society for 1947-1948, were handed to the Secretary in accordance with the By-Laws:

President-Elect—Dr. Max M. Green. Endorsed by Drs. I. Tedesco, Jules Myron Davidson, N. J. Tessitore and Daniel J. Murphy.

First Vice-President—Dr. J. Kelly Stone. Endorsed by: Drs. Donovan C. Browne and George Hauser.

Second Vice-President—Dr. C. J. Brown. Endorsed by: Drs. Joseph P. Palermo and Max M. Green.

Third Vice-President—Dr. J. O. Weilbaecher, Jr. Endorsed by: Drs. Wm. H. Roeling and Eugene H. Countiss.

Secretary—Dr. N. J. Tessitore. Endorsed by: Drs. N. J. Chetta and C. Barrett Kennedy.

Treasurer—Dr. Boni J. DeLaureal. Endorsed by: Drs. Eugene H. Countiss and C. Grenes Cole.

Librarian—Dr. Eugene H. Countiss. Endorsed by: Drs. Donovan C. Browne and Eugene B. Vickery.

Additional Members Board of Directors—Dr. E. L. Leckert. Endorsed by: Drs. J. G. Menville, L. A. Delery, L. J. Gehbaeur and Jos. J. Ciolino. Dr. Charles B. Odom. Endorsed by: Drs. Val Fuchs and E. J. Richard.

DELEGATES TO THE LOUISIANA STATE MEDICAL SOCIETY

Dr. Ruth Aleman. Endorsed by: Drs. N. J. Chetta and Vincent P. Blandino.

Dr. Boni J. DeLaureal. Endorsed by: Drs. Edwin L. Zander and Vincent P. Blandino.

Dr. P. Graffagnino. Endorsed by: Drs. B. Hauser, Max M. Green and John F. Oakley.

Dr. Max M. Green. Endorsed by: Drs. Frank Chetta and Wm. H. Roeling.

Dr. George Hauser. Endorsed by: Drs. John T. O'Ferrall and E. J. Richard.

Dr. John Menville. Endorsed by: Drs. Jules Myron Davidson and N. J. Tessitore.

Dr. Waldemar Metz. Endorsed by: Drs. E. L. Leckert and L. S. Charbonnet.

Dr. Daniel J. Murphy. Endorsed by: Drs. Ignatius Tedesco and H. Ashton Thomas.

Dr. J. D. Rives. Endorsed by: Drs. Joseph P. Palermo and C. J. Brown.

Dr. Robert Sharp. Endorsed by: Drs. John Menville and Donovan C. Browne.

Dr. Edmond Souchon. Endorsed by: Drs. Eugene H. Countiss and Pascall L. Danna.

Dr. N. J. Tessitore. Endorsed by: Drs. E. A. Fatter and L. C. Delery.

Dr. Eugene B. Vickery. Endorsed by: Drs. John F. Oakley and Edward Matthews.

Since there is no opposition the Secretary will cast the ballot for the Society on Saturday, December 14, 1946.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of very month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

RAPIDES PARISH MEDICAL SOCIETY

The Rapides Parish Medical Society held its regular monthly meeting at the Hotel Bentley on Monday, November 4. Speaker of the evening was Dr. H. Whitney Boggs of Shreveport who spoke on the interesting subject of new developments in the treatment of the allergic patient of particular interest to the general practitioner.

SIXTH DISTRICT MEDICAL SOCIETY

The Sixth District Medical Society held its Fall Meeting in Plaquemine, Louisiana, on Thursday, November 7, 1946.

Dr. Charles J. Bloom read a paper on "Recent Advance in Pediatric Practice". Drs. Gilbert Anderson and Andrew Friedricks held a "Clinical-Pathological Conference". Dr. Will E. Barker Jr., President, presided at the meeting. There was a very good attendance.

ASCENSION PARISH MEDICAL SOCIETY

A regular meeting of the Ascension Parish Medical Society was held at the Gonzales Hospital on Thursday, October 31, at 7:30. The following were present: Drs. Henry Folse, Percy Le Blanc, Earl Schexnayder, Bertha Stokes, D. C. Brumfield, E. S. Kyes, Guy St. Amant, Alvin St. Amant, Myer Epstein, Elsie B. Cassidy, Maurice McConnel, and G. L. Gaudin, Dr. Silverman the guest speaker, and Dr. Alan Leslie, of New Orleans.

It was decided by the society that the regular meeting in December be a joint meeting with the election of officers and scientific program. The society went on record as endorsing the action of the State society in regard the E.M.I.C. program. Drs. Maurice McConnel, Alvin St. Amant, and Elsie B. Cassidy were voted in as new members of the society and Drs. Silverman and Leslie as honorary members.

A paper entitled "The Unstable Colon" was presented by Dr. Earl Schexnayder. Drs. Percy Le Blanc and J. M. McConnel gave brief discussions of this paper and Dr. Silverman, a noted authority on the subject, discussed the paper.

Following the adjournment a most delicious steak supper was served at the new Airliner Restaurant.

G. L. Gaudin, M.D., Sec. Treas.

THE TOURO MEDICAL STAFF MEETING

The regular monthly meeting of the Medical Staff of Touro Infirmary was held Wednesday, November 13, at which time the following program was presented:

First came a Clinico-Pathologic Conference led by the Department of Pathology, the clinical discussion of the case being made by Irving J. Glassberg.

The next scientific presentation was a paper by Drs. E. Burns and W. E. Kittredge on ureterocutaneous anastomosis.

The program was concluded by a 15 minute sound film on sulfathalidine in the treatment of ulcerative colitis.

The Program Committee of the Staff consisted of Drs. Sidney Copland, Oscar Blitz, and Harry Meyer.

SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE (SOUTHERN SECTION)

Dr. Chapman Reynolds has sent out notices from the Department of Pharmacology of the L. S. U. School of Medicine to the effect that the next scientific meeting of the Society will be held in Birmingham on December 7. He suggests in his notice that car-pools be established in Memphis, Atlanta, and New Orleans so that members might drive to this meeting together.

MEDICAL DIVISION OF THE CHARITY HOSPITAL VISITING STAFF

The regular monthly meeting was held at the Auditorium of the Charity Hospital, November 19. The program consisted of a paper by Dr. N. Burnstein on Boeck's Sarcoid. The discussion of this paper was opened by Dr. Chester Fresh. A medical case was presented by Dr. C. B. Luikart which was discussed by Dr. Seabury. There was a dermatology case presentation by the members of the Louisiana State University Staff.

The Officers of this Section for the coming year are Dr. W. Sodeman, Chairman, Dr. E. S. Kagy, Vice-Chairman, and Dr. J. Wells, Secretary.

PHYSICIANS ELECTED BY TULANE ALUMNI

Dr. Walter L. Moss of Lake Charles was selected as president of the Tulane Alumni Association when this organization held its annual meeting on Saturday, November 23. Dr. Moss graduated from the Medical School of Tulane in 1927 and when an undergraduate student was president of the Medical School student body and the University student council.

At this same meeting Dr. Ambrose Storck was elected a member of the Executive Committee and Dr. Charles Odom was selected to fill a vacancy on the Committee.

NEW HEAD OF ANATOMY DEPARTMENT AT L.S.U. MEDICAL SCHOOL

Dean Vernon Lippard has announced the appointment of Dr. Charles M. Goss as professor and head of the Department of Anatomy at Louisiana State University Medical School. Dr. Goss is well known to anatomists. At the present time he is head of the Department of Anatomy at the University of Alabama. It may be of interest to the medical profession to known that Dr. Goss is preparing a new edition of Gray's Anatomy. This standard textbook on anatomy has been published for many decades. It is probably the oldest book on any medical subject that is in active use. It has been said of Gray's Anatomy that more copies of it have been sold than any other medical book with the possible exception of Sir William Osler's Principles and Practice of Medicine.

ANNUAL MEETING OF THE FELLOWS OF THE AMERICAN COLLEGE OF SURGEONS

In accordance with Article III, Section 5, of the By-Laws, the annual meeting of the Fellows of the American College of Surgeons is called for 1:45 o'clock on the afternoon of Thursday, December 19, 1946, in the Music Hall, Public Auditorium, Cleveland, Ohio.

This meeting constitutes the annual business meeting of the Fellows of the College, at which time officers are elected and reports from officials of the College are presented. Attached is the program for the meeting.

Each Fellow of the College is respectfully urged to be present.

Irvin Abell, M.D.,
Chairman, Board of Regents

A most interesting scientific program has been arranged for this Annual Convention of the American College of Surgeons. There will be a forum on fundamental surgical problems four days in the week. There will be evening symposia in general surgery on Tuesday, Wednesday and Thursday nights and at the afternoon panel discussion which will be held Monday through Friday afternoons, there are surgical problems to be presented.

The technical exhibit, together with the registration and clinic ticket bureaus, will be held in the arena of the Cleveland Public Auditorium.

REGIONAL MEETING OF THE AMERICAN COLLEGE OF PHYSICIANS

A regional meeting of the American College of Physicians was held in Memphis on November 22, honoring Dr. Hugh Morgan, president-elect of the College. The committee in charge included governors from Texas, Arkansas, Tennessee, Mississippi and Louisiana, the latter being Dr. Edgar Hull, Professor of Medicine at Louisiana State University Medical School.

FELLOWSHIPS IN CANCER RESEARCH

Fellowships in cancer research of the American Cancer Society which is administered by the Committee on Growth of the National Research Council, are available to those who wish advanced training and experience in any field of investiga-

tion pertaining to the problems of cancer. These fellowships if on a junior basis pay from $2,000 to $3,000 a year; for the senior fellowships the stipend is from $3,000 to $6,000 per annum. The Committee on Growth reserves the right to select the institution in which the fellowship is to be applicable.

THE SOUTHERN MEDICAL ASSOCIATION MEETING

At the recent meeting of the Southern Medical Association at Miami, Florida the first part of November, Dr. Gordon McHardy was selected to be chairman of the section on gastro-enterology, Dr. Ralph V. Platou, chairman of the section on pediatrics, and Dr. Merrill C. Beck, chairman of the section on anesthesiology, succeeding Dr. John Adriani.

The following Louisiana doctors were to appear on the program either to read papers or open discussions: Drs. William A. Sodeman, Thomas Findley, Rawley Penick, Jr., Grace A. Goldsmith, Harold Cummins, Ralph V. Platou, M. T. Van Studdiford, Lee D. McLean, Arthuro Alvarado, Russell Holman, James H. Eddy, Jr., Lyon K. Loomis, Thomas Benton Sellers, W. R. Buffington, William B. Clark, George M. Haik, David A. Davis, John Adriani, Maxwell E. Lapham and John H. Whitney, all of New Orleans; C. H. Webb, S. George Wolfe, R. T. Lucas, Charles E. Anderson, Shreveport, and Fritz LaCour, Lake Charles.

Drs. Grace Goldsmith, Bernard G. Efron, George Burch, Thorpe Ray, J. Brown Farrior, Neal Owens and John Adriani had scientific exhibits.

Mrs. A. A. Herold of Shreveport presided over the sessions of the Women's Auxiliary and Dr. Ruth G. Aleman was chairman of the women physicians group.

The following members of the State Medical Society were officially registered at the convention:

Drs. Maunsel B. Pearce, Monroe B. Wilson, and Allen C. Winters, of Alexandria, Drs. Guy H. Faget and F. A. Johansen, Sr., of Carville, Dr. Jos. H. Pankey of Ferriday, Dr. Pegram L. McCreary of Lake Charles, Dr. George W. Wright of Monroe, Drs. Woodward D. Beacham, Louis A. Breffeilh, Edgar Burns, Guy A. Caldwell, O. P. Daly, Jos. S. D'Antoni, Roy E. de la Houssaye, Ernest Carroll Faust, Geo. D. Feldner, John E. Garcia, C. S. Holbrook, Edgar Hull, Leo Francis Klenk, E. L. King, Edwin H. Lawson, Lucien A. LeDoux, Francis E. LeJeune, Lodwick S. Meriwether, Isidore L. Robbins, Robert. A. Robinson, Jr., Daniel H. Rowe, R. F. Sharp, Henry Ashton Thomas, H. R. Unsworth, Arthur J. Walker, and Monroe Wolf, of New Orleans, Dr. Henry S. Roans of Ruston, Dr. A. A. Herold of Shreveport, Dr. Rosamond R. Arceneaux of Welsh.

We were delighted to learn that Dr. Lucien LeDoux was elected president of the organization and that Mrs. Wiley Buffington was elected president of the Women's Auxiliary. Editorial Comment on these two elections will appear later.

OCHSNER RECEIVES TIMES PICAYUNE LOVING CUP FOR 1945

The Times Picayune announced in its November 17 edition that Dr. Alton Ochsner had been awarded the Times Picayune Loving Cup for cumulative community service. He was selected by a committe composed of representatives from the Association of Commerce, the Board of Trade and the Cotton Exchange.

The award of this loving cup to Dr. Ochsner is well merited. He has been an outstanding man in this community since he became Professor of Surgery at Tulane in 1926.

Dr. Ochsner is the second physician to receive this honor. In 1940 the loving cup was presented to Dr. Rudolph Matas.

AN IMPORTANT RESOLUTION

The following resolution was unanimously approved and passed at the Twenty-Eighth National American Legion Convention, in San Francisco, October 4, 1946.

Whereas: Veterans who have served in the armed forces now have available to them hospital and medical care provided by the United States Government; and

Whereas: There are countless voluntary health insurance plans now being offered by the physicians and the insurance companies; and

Whereas: Proposed plans of compulsory health insurance would increase the tax burden and bring about regimentation of the medical profession; and

Whereas: All forms of compulsion are repugnant to our American way of life since our liberties and opportunities would be circumscribed;

Now, Therefore, Be It Resolved: That the National Assembly of the American Legion hereby expresses its opposition to compulsory health insurance.

We are deeply indebted to Dr. Joseph C. Menendez who headed the Delegation from Louisiana to the American Legion meeting in San Francisco and to the delegates themselves for their cooperation in supporting the resolutions opposing compulsory health insurance.

Dr. Daniel Murphy, President of the Orleans Parish Medical Society, has announced approval by the Society of the plans to establish an Eye-Bank at the Eye, Ear, Nose and Throat Hospital. Approval was subject to the medical profession being in charge of all medical decisions.

This Eye-Bank will serve the Southeastern area, the eyes being transported in cooperation with the Eastern Air Lines and the Red Cross Motor Corps.

PLAUCHE HOSPITAL ASSIGNED TO OCHSNER FOUNDATION

Announcement was made approximately about the middle of November that the Plauche Hospital had been turned over to the Ochsner Foundation. Final consummation of the contract with the government will probably be completed within the next week and officially the institution will be under civilian control. The new hospital will be known as the Foundation Hospital and will be open not only to members of the Ochsner Clinic, but also to any member of the Orleans Parish Medical Society. It is expected that the painting, redecorating and rearrangement of the buildings will be completed within the next two months and that the hospital will open formally for the care of patients on February 1.

NEWS ITEMS

Dr. Mercer G. Lynch has been nominated by the Secretary of War as a Civilian Consultant to the Secretary of War in his special field of otolaryngology.

Dr. Edward L. Bortz, chairman of the Committee on National Emergency Medical Service of the American Medical Association, has announced that there will soon be mailed a comprehensive questionnaire to more than 45,000 discharged medical officers of World War II. It is urged that all medical officer veterans answer this questionnaire as promptly as possible so that it may serve as a guide in preparing for any new national emergency.

The United States Public Health Service recently dedicated the eighth building of the National Institute of Health at Bethesda, Maryland. This Memorial Laboratory will be devoted to research on the Rickettsial diseases, tularemia, psittacosis, poliomyelitis, and other central nervous virus diseases.

At Washington on November 7, twenty-four representatives met with the National Advisory Cancer Council to discuss cancer from the angle of medical education. This meeting was held under the auspices of the National Cancer Institute of Bethesda.

Representing Louisiana were Dr. M. E. Lapham of the school of medicine at Tulane and Dr. Alton Ochsner, professor of surgery at Tulane.

Over $100,000,000 has been paid by the United States Government under the Emergency Maternity and Infant Care Program. At the peak between 40 and 50,000 cases have been authorized a month. Now the authorization averages about 15,000 a month. In the state of Louisiana there were 15,200 births under the E.M.I.C. Program.

Dr. J. Brown Farrior was awarded the second prize for his scientific exhibit on the ear at the recent meeting of the Southern Medical Association.

Dr. Grace Goldsmith of the Department of Medicine at Tulane and Drs. Burch and Ray of the same department received honorable mention for their exhibits.

AMERICAN UROLOGICAL ASSOCIATION

"*Urology Award*—The American Urological Association offers an annual award 'not to exceed $500' for an essay (or essays) on the result of some clinical or laboratory research in urology. Competition shall be limited to urologists who have been in such specific practice for not more than five years and to residents in urology in recognized hospitals.

For full particulars write the Secretary, Dr. Thomas D. Moore, 899 Madison Avenue, Memphis, Tennessee. Essays must be in his hands before May 1, 1947.

The selected essay (or essays) will appear on the program of the forthcoming meeting of the American Urological Association, to be held at the Hotel Statler, Buffalo, New York, June 30-July 3, 1947".

Committee on Scientific Research
Miley B. Wesson, Chairman
Judson B. Gilbert
Anson L. Clark.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY, INC.

The next written examination (Part I) for all candidates will be held in various cities of the United States and Canada on Friday, February 7, 1947, at 2:00 p. m. Candidates who successfully complete the Part I examination proceed automatically to the Part II examination to be held in Pittsburgh, Pa., June 1-7, 1947. All candidates, especially those in military service, are requested to keep the Secretary's Office closely informed of changes in address.

Several changes in Board regulations and requirements were put into effect at the last annual meeting of the Board held in Chicago, Illinois, from May 5 to May 11, 1946.

For further information and application blanks address—Paul Titus, M.D., Secretary, 1015 Highland Building, Pittsburgh, 6, Pennsylvania.

MISSISSIPPI STATE MEDICAL EDUCATION PROGRAM

The Mississippi State Medical Education Board announces the granting of 47 scholarship loans effective the first semester of 1946-47. These loans

have been awarded to young men and women from 28 towns in 23 counties in Mississippi. They will attend different medical schools, many of them taking their preclincal work at the University of Mississippi Medical School, a two-year institution. The students in the clinical branches will attend 11 different medical schools throughout the nation. The majority of these will go to Tulane University, with Louisiana State University and the University of Tennessee Colleges of Medicine having an equal number.

The action taken by the Mississippi legislature in granting the scholarship loans to prospective medical students and doctors is, we believe, one of the most outstanding advances in the medical education that has been consummated in recent years. It is to be borne in mind that medical schools can only be established satisfactorily in large centers of population and there are very few states in this country that have sufficiently large hospitals for the adequate training of medical students. It would be very much better for many more were schools to follow the Mississippi plan than it would be to attempt to carry on with a lack of clinical material and with a faculty which will show marked weaknesses in certain divisions. Notably those are the clinic divisions because there are not enough well-trained physicians in the locality who are interested in training students and devoting time to the teaching hours that are required.

FELLOWSHIPS IN MEDICAL RESEARCH

The Surgeon-General of the United States Public Health Service, Dr. Thomas Parran, has announced that approximately 120 one-year fellowships in medical research are open to graduate science students. The details of these research fellowships may be obtained from Dr. Dyer, Director of the National Institute of Health, Bethesda, Maryland.

NOVEMBER ISSUE WANTED

Doctors who do not wish to retain copies of the New Orleans Medical and Surgical Journal are requested to return copies of the November, 1946 issue to the office of the Journal due to a shortage of extra copies of this issue.

HEALTH IN NEW ORLEANS

The Bureau of the Census, Department of Commerce, reported that for the week ending October 5 there were 141 deaths in the city of New Orleans, divided 93 white and 48 colored. Of these deaths 21 were children under one year of age, 15 white and six colored. These figures were considerably higher than for the last week reported which ended September 28 in which there were 122 deaths. The following week, which closed October 12, there was a marked diminution in the number of deaths that took place in the city. In this week there were 72 white people who died, 35 colored, with 15 of the deaths occurring in children under one year of age. Strange to say, 13 of these deaths were in white children which is quite unusual. For the following week, terminating October 19, the death rate jumped materially because of the death of 142 of the citizenry of the city, of whom 95 were white, 47 were colored, with only 11 deaths in children under one year. There seems to be rather a marked fluctuation in the number of death in the city from week to week; for that which closed October 26, there was a considerable fall with 119 deaths reported, 78 of which were white and 41 were colored, with 13 children dying this week, eight of whom were colored. The week which came to an end on November 2 presented figures almost identical with the previous week. There was one fewer deaths than the week before. Of the 118 people who died in the city, 75 were white, 43 were colored and ten were children under one year old.

INFECTIOUS DISEASES IN LOUISIANA

The weekly morbidity report for the State of Louisiana for the week ending October 5, showed the following reportable diseases in numbers greater than ten; cancer 173, pulmonary tuberculosis 122, unclassified pneumonia 111, hookworm infestation 14 and poliomyelitis 11. The poliomyelitis cases were reported throughout the state with no one parish having more than three cases originating within its boundaries. There was a rather marked difference from the previous week the following week in the number of reported cases, there being only three occurring in numbers greater than ten, these being pulmonary tuberculosis 63 cases, hookworm infestation 21 and typhus fever 16. There were nine cases of poliomyelitis reported this week, again the cases being scattered throughout the state. Of the large number of typhus fever cases, 16 in all, eight of them were reported from Orleans Parish. For the week which closed October 19, pulmonary tuberculosis again led all reportable diseases with 43 cases, followed by 37 of malaria, 29 of influenza, 20 of septic sore throat, and 14 of hookworm disease. Thirty-five cases of malaria were reported from Jackson Parish. Seven cases of poliomyelitis were reported this week, three of which came from Tangipahoa Parish and two from Orleans. The week which ended October 26 was an unusually healthy week, there being reported only three diseases in numbers over ten, these being hookworm infestation 25, pulmonary tuberculosis 21 and scarlet fever 13. The seven cases of poliomyelitis that were listed developed in six different parishes widely scattered over the state.

MONTHLY MORBIDITY FOR VENEREAL DISEASES, STATE OF LOUISIANA

Month Ending September 30, 1946

	Total This Month	Total Previous Months	Total To Date 1946
Chancroid	49	578	527
Gonorrhea	1246	13257	14503
Granuloma Inguinale	22	219	241
Lymphopathia Venereum	8	83	91
Syphilis	1580	13760	15340

NAVAL AIR RESERVE TRAINING COMMAND
U. S. NAVAL AIR STATION
GLENVIEW, ILLINOIS

Secretary Louisiana State Medical Society
1430 Tulane Avenue
New Orleans, Louisiana
Dear Doctor:

Attached herewith is a rewrite of the need of Naval Medical Officers in the Naval Air Reserve Training program.

Circulation of the contents of this notice among the members of your State organization will be sincerely appreciated as it is believed that many Ex-Naval Medical Officers would be desirous of continuing their association with the Navy on a full time basis under conditions outlined or to a limited extent with the Naval and Marine air groups of the Naval Reserve in regular periods of training duty.

Thanking you in advance for your cooperation.

Yours very truly,
G. B. Ribble,
Captain (MC) USN
Staff Medical Officer

ATTENTION NAVAL RESERVE OFFICERS!

Full Time Active Duty: The attention of Reserve Medical Officers and of Pharmacists of the Hospital Corps is invited to the opportunity to perform *full-time active duty* at one of the major naval air stations of the Naval Air Reserve Training Command or at one of the Naval Air Reserve Training Units (NARTUs) listed below.

Stations of the NARTC

NAS, Atlanta, Ga.
NAS, Columbus, Ohio.
NAS, Dallas, Texas.
NAS, Glenview, Ill.
NAS, Grosse Ile, Mich.
NAS, Los Alamitos, Cal.
NAS, Memphis, Tenn.
NAS, Minneapolis, Minn.
NAS, New Orleans, La.
NAS, New York, N. Y.
NAS, Oakland, Cal.
NAS, Olathe, Kan.
NAS, Squantum, Mass.
NAS, St. Louis, Mo.
NAS, Willow Grove, Pa.

NARTUs of the NARTC (Based at the below listed Naval Air Stations)

Anacostia, D. C.
Jacksonville, Fla.
Miami, Fla.
Norfolk, Va.
San Diego, Cal.
Seattle, Wash.

How to Apply: Officers in the above category who are interested in *full-time active duty* as a member of the stationkeeper staff at one of the stations or units listed above should initiate letters to the Bureau of Naval Personnel, via the Chief of Naval Air Reserve Training, Naval Air Station, Glenview, Ill., and BuMed, listing three or four stations at which duty is desired in order of preference. Personnel are desired in the ranks of Commander and Lieutenant Commander in the Medical Corps.

Advantages: Officers qualifying for the above billets can be assured of every consideration in obtaining and continuing duty at the station of their preference. It is the policy of the Command to obtain "orders to duty involving flying" for designated Naval Flight Surgeons. Government quarters are available at many of the major Naval Air Stations.

Part Time Duty: Naval Flight Surgeons of the Reserve who are desirous of affiliating themselves with either the Organized or Volunteer components of the Inactive Reserve composed of Naval or Marine air groups training at one of the Naval Air Stations or NARTUs listed above should contact the Commanding Officer of the station or the NARTU at which the training unit is based.

Do not miss this splendid opportunity to enjoy an indefinite tour of "shore duty only" in a peace time Navy at one of the Naval Air Stations listed above, thereby both benefiting yourself and the Navy by contributing your services to the post-war Naval Air Training Program!!

BROOX CLEVELAND GARRETT, M.D.
1888-1946

Dr. B. C. Garrett, of Shreveport, died on November 15, 1946, after a protracted illness. In the passing of Dr. Garrett many medical men throughout the state will lose a good and loyal friend. He was universally popular and very generally liked by every one.

Dr. B. C., as he was generally known, graduated from Tulane in 1913 and two years later became a member of the State Medical Society, holding active membership until elected an honorary member in 1946. He was a college athlete of renown and all his life his chief avocation with connected with outdoor activities; hunting, shooting and farming, but to Dr. Garrett there was not much time for play as after entering practice he rapidly developed one of the largest surgical practices in

Shreveport which he maintained up until the time of his last illness. In the death of Dr. Garrett a great many of us have lost a real and true friend, a man who could be depended upon in times of difficulty and who was always willing to help not only his friends but all others.

LESLIE LEON LUMSDEN
1875-1946

One of the outstanding leaders in the development of public health in the United States, Dr. Leslie L. Lumsden, died November 8 at the United States Marine Hospital, New Orleans. His death at the age of 71 was due to leukemia complicated by pneumonia.

From the time that Dr. Lumsden was commissioned an Assistant Surgeon in 1898 until he retired in 1939, he consistently shattered the accepted beliefs of his profession. By tireless research and field investigations, Dr. Lumsden exposed true vectors of disease, definitely established sources of contamination in the community, and originated and developed full time rural health plans.

He uncovered all the facts concerning the spread of typhoid fever by means of unsanitary food and water as well as flies and other carriers. The reports of his investigations served as the basis of future control measures for the disease.

BOOK REVIEWS

Early Ambulation and Relative Procedures in Surgical Management: By Daniel J. Leithauser, M. D., F. A. C. S. Springfield, Illinois, Charles C. Thomas, 1946. Pp. 232. Price, $4.50.

This volume excites interest and emotion without necessarily stimulating enthusiasm.

The easy complacency of the author in regard to his own attitudes and his assumption that the opinions contrary to his own are based on ignorance and tradition, is to say the least interesting.

This small volume of two hundred odd pages is a compendium of the author's interpretation of physiology, anatomy, surgical technic, surgical nursing, and testimonials from patients.

I fear that the author has not given just credit to surgeons whose procedures for preventing postoperative complications and disabilities differ from his own. In all matters controversial, time and judgment of the majority will provide an adequate answer.

ISIDORE COHN, M. D.

Jewish Luminaries in Medical History: By Harry Friedenwald, M. D., D. H. L. (Hon.), D. Sc. (Hon.). Baltimore, The Johns Hopkins Press. 1946. Pp. 199. Price, $3.00.

This volume has many distinguishing characteristics. It is a choice morsel from the pen of the greatest living student of that phase of medical history which has to do with the part played by Jews in the development of medical practice.

Dr. Friedenwald's monumental, epoch making, two volume work on "The Jews and Medicine" was published in 1944 by The Johns Hopkins Press.

Dr. Friedenwald gives due credit for his interest in medical history to Osler, Welsh, and his own father, the late Dr. Aaron Friedenwald. By this tribute Friedenwald glorifies the work of his preceptors, and by his work his name is added to the list of great medical historians.

Sigerist states in the foreword that Friedenwald is a great book collector. He adds "Unlike other collectors, he reads his books, studies them, and uses them as historical sources."

As a witness to the truth of this statement, one has but to turn to the catalog (which forms part of this volume)—"Of works bearing on the subject of the Jews and Medicine from the private library of Harry Friedenwald," to realize that in his library may be found the finest collection on the subject in the United States, and, I would venture, probably the equal of any in the world.

Students of medical history will find a library incomplete without the two volume work of Friedenwald's on "The Jew in Medicine" and this essay on "Jewish Luminaries in Medical History" along with the catalog of Friedenwald's library.

ISIDORE COHN, M. D.

Pneumoperitoneum Treatment: By Andrew Ladislaus Banyai, M. D., F. A. C. P., F. C. C. P. St. Louis, C. V. Mosby Co., 1946. Illus. Pp. 376.

An authoritative monograph on the pneumoperitoneum treatment of pulmonary tuberculosis is quite timely in view of the present widespread interest in this method. The author of this book, both by priority and by sustained interest in the subject, is probably the best qualified man in this country to do it full justice. He has covered it exhaustively from the introductory historical review to the bibliography of almost 700 references (including 24 of his own papers on pneumoperitoneum). Everything possibly related to pneumoperitoneum is there. Therefore the result is a reference work rather than a practical manual to guide the physician who wishes to employ this form of treatment. The 32 pages devoted to technic of administration will be found useful as will also the 130 pages on the treatment of pulmonary tuberculosis. Some question might be raised as to the validity of

the physiologic concepts in the discussion of intra-abdominal pressure. This method in the treatment of all the conditions covered in the other ten chapters on therapeutic application is at best of doubtful utility. The illustrations are numerous and adequate to show the gross roentgenologic features of the method although finer details are lost in the reproduction. This monograph may profitably be referred to by anyone interested in the treatment of tuberculosis or other pulmonary diseases. However it should be read with the realization that it is not a critical analysis of a method of treatment which is still under trial and must always be supplementary to more basic forms of therapy.

J. L. WILSON, M. D.

Peptic Ulcer, its Diagnosis and Treatment: By I. W. Held, M. D., F. A. C. P. and A. Allen Goldbloom, M. D., F. A. C. P. Springfield, Ill., Chas. C. Thomas, 1946. Pp. 382. Price, $6.50.

This small concise volume of three hundred and eighty-two pages lives up to the authors' claim that it is a clinical treatise on the diagnosis and treatment of peptic ulcer. The contention that it is suited for the practitioner is probably true; the reviewer was not impressed with its applicability to the physician in the military service; the student can get as much information from the average endorsed text book on internal medicine. It is apparent that I am not impressed that this is an authoritative treatise on peptic ulcer.

In the section on pathogenesis the authors indifferently enumerate the possibilities, briefly treat on the all important neurogenic factors and completely omit any reference to the experimental production of peptic ulcer by histamine.

The chapter on pathology is not impressive.

In consideration of symptomatology proper emphasis is given to the periodicity of pain.

A relatively too elaborate consideration is given the differential diagnosis of a rather clear cut clinical syndrome applicable to a perforated ulcer.

The good point on the consideration of hemorrhage is that persistent severe bleeding probably indicates pancreatic duodenal vessel erosion.

In the management of stenosis the authors are extremely vague as to where the internist gives up to the surgeon. Again the same vagueness exists as to when to elect surgery in a gastric ulcer not responding to medical measures. Like so many other authors here again too much emphasis is placed on the site of an ulcer as to its malignant potentialities when every good clinician knows carcinoma can occur in any portion of the stomach.

The indications for surgery are well outlined. The types of operative procedure are handled too indifferently even for an internist. No mention is made of vagotomy nor of jejunal transplants.

The therapeusis is poorly and indifferently presented with the opening statement that there is "no specific treatment". One can not manage ulcer patients well with such an attitude, there may be no specific treatment but there is an acceptable method of management of disease and patient. The mention of such discarded products as Larostidin and Synodal without giving an evaluation of their efficacy is not commendable.

The reviewer is interested to know there is a "flexible esophagoscope".

The entitlement Gastric Catarrh is meant to indicate the established entity gastritis. The chapter is extremely poor, does not approach a comprehensive knowledge of the subject and omits the essential feature to the entity, i.e., the gastroscopic evaluation.

A chapter dealing with the "dyspeptic soldier" insults him by failing to conclude the possible organic background for his symptomatology, without admitting the inadequacies of many military examinations and follow-up studies.

The portion dedicated to roentgen evaluation seems adequate. It is surprisingly well done when one considers this in contrast to the mention of the existence of gastroscopy and the absence of mention of gastrophotoscopy. One finds figures No. 27 and No. 33 reversed.

I wonder if many clinicians consider that the manifestations of a peptic ulcer call into consideration for differential diagnosis such entities as: mucus colitis and dibothrophillin latum infestation; the authors do.

While interesting themselves too surgically in the immediate postoperative management the authors neglect to consider the many physiologic disturbances that occur as to result of surgery.

I am not inclined to endorse this volume as an authoritative treatise on the diagnosis and treatment of peptic ulcer. It is well arranged, easily readable and is adequately illustrated.

GORDON MCHARDY, M. D.

PUBLICATIONS RECEIVED

The Dial Press, New York: The Challenge of Polio, by Roland H. Berg.

Grune & Stratton, New York: Clinical Methods of Neuro-Ophthalmologic Examination, by Alfred Kestenbaum, M. D.

Mead Johnson & Company, Evansville, Indiana: Courage and Devotion Beyond the Call of Duty. A Manual for the General Practitioner.

Henry Schuman, New York: Memoir on Sulphuric Ether, by W. T. G. Morton. Victory Over Pain, by Victor Robinson, M. D. The Centennial of Surgical Anesthesia by John F. Fulton, M. D., and Madeline E. Stanton, A. B. Journal of the History of Medicine and Allied Sciences, a Quarterly.

Commonwealth Fund, New York: Dentistry, An Agency of Health Service, by Malcolm Wallace Carr, D. D. S.